

مُذَكِّرات

طبيب امتياز

طبيب: مُحَمَّد بركات

مدير منتديات كل الطب








طبيب امتياز

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1st Edition

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إهداء

أحمد الله أن وفقني لإتمام هذا العمل

وأسأله أن يكون خالصا لوجهه

وأن يكون سببا لنفع كل زملائنا وكل المرضي إن شاء الله.

أهدي هذا الكتاب لأبي وأمي وزوجتي وطفلتي مريم وكل أهلي وكل من ساعدني

وأدعوا الله لهم جميعا بأن يرضي عني وعنهم وعن كل المسلمين.

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مقدمـة

السلام عليكم ورحمة الله وبركاته

إن الحمد لله نحمده ونستعين به ونستهديه ونستغفره ونعوذ بالله من شرور أنفسنا ومن سيئات أعمالنا

اللهم علمنا ما ينفعنا وانفعنا بما علمتنا واجعل كل أعمالنا خالصة لوجهك الكريم ولا تجعل لأحد غيرك فيها شيئا

أما بعد

هذه مذكراتي أكتبها لزملائنا أطباء الامتياز ولزملائنا في كلية الطب البشري .. شرعت في كتابتها وجائتني فكرتها في أواخر الامتياز ولم يكن هناك نية لها من البداية لكني عزمت علي كتابتها والانتهاء منها سريعا ليستفيد منها الجميع

وما دفعني حقيقة لكتابتها هو ما وجدته من الكثير من الأخطاء الطبية والمهنية للكثير من الزملاء ولا أحد يحاول تصحيح خطأه او يصلح من نفسه أو يزداد علما.. وما دفعني أيضا أن كل الكتب تقريبا الموجودة علي الساحة الخاصة بأطباء الامتياز تقدم عبارة عن معلومات جاهزة مشكور من كتبها لكنها لا تربي فينا حب المعرفة وحب الاطلاع والاعتماد علي النفس .. فقد تكون هناك معلومة خاطئة أو ناقصة .. فأرد أن أقدم لكم أسلوبا جديدا في الكتابة يستفيد منه الجميع ويغرس فيهم بعض الأمور الهامة كما سأكتبها لاحقا

قد لا أكون أحسن الكتابة لكني أضمن لك أن تحصل علي المعلومة وترسخ في ذهنك عن طريق الأسلوب الذي سرت عليه وهو أسلوب الحالات الطبية بدلا من سرد المعلومات .

وجدت الكثير من زملائنا في الامتياز يعتمد علي الكتب التي تقدم الحلول السريعة .. بمعني أصح ساندوتش سريع للمعلومة وللموضوع . وهذا يصلح قبل الامتحانات من مذكرات ما قبل الامتحان ((مع احترامي الكامل لكل المذكرات وكل كتابها فأنا استفدت منهم شخصيا)) لكني وجدتهم تقريبا مقصرين فيما يقدمونه لكم خصوصا ترسيخ الأفكار التي سأوضحها.

تخرجنا من كلية الطب بعد معاناة سنوات متواصلة وخضنا المئات من الامتحانات وحشونا رأسنا بملايين المعلومات التي نسينا نصفها أو لأكون منصفا تم تخزينها في الذاكرة البعيدة لكنها تحتاج جهدا كبيرا لإزالة التراب من عليها وإعادة صياغتها بما يتماشي مع المرحلة الجديد وهي مرحلة الاحتكاك بالمريض بدلا من الاحتكاك بالكتاب فقط

طبيب الامتياز لا يحتاج معلومات جاهز بل يجب عليه أن يبحث في الموضوع ويقرأ فيه كل صغيرة وكبيرة وكل جديد فيه لأنه أصبح من الان يتعامل مع مريض يتعامل مع نفس بشرية عليه احترامها واحترام حقوقها ومن حقه أن يتم تشخيصه وتقديم العلاج اللازم له والشفاء علي الله عز وجل لأن الله هو الطبيب .. الله هو الشافي .. بينما نحن سبب في الشفاء .

ستجدوني في هذه المذكرات أقدم لكم حالة عشتها وتعايشت معها بكل تفاصيلها .. ثم في المقابل شخصت لكم الحالة وأعطيتكم مفاتيح لها .. وقدمت لكم الموضوع كاملا للقراءة والمذاكرة والبحث من مصادر علمية موثقة .. وقدمت لكم أساليب عن طريق صور من مصادر طبية تساعدكم في كيفية التعامل مع الحالة وكيفية التفكير فيها.

ولن تجدني في هذه المذكرات قد كتبت لكم الدواء والروشتة السحرية التي ستأتي بالنتيجة مع المريض فأنا لم أعتمد في حياتي المهنية القصيرة علي هذه الروشتات .. بل اعتمدت علي ما كتب في المراجع العلمية من أهل العلم الثقة وبحثت عن الأدوية بنفسي وأتيت بأدوية وجدت أن الكثيرين لم يكتبوها أو لم ينوهوا عنها لذلك لن تجدني أكتب في نهاية الموضوع الدواء التجاري وووووو

هذه أمور ينبغي عليكم أن تبحثوا فيها وصدقوني ستجدون المتعة في ذلك وستجدون أنكم تفهمون المرض وأن علاجه يحتاج كذا وكذا وبالتالي يجب كتابة كذا وكذا وهنا يأتي دور أطلس الأدوية الذي سيكون معكم لتعرفوا المادة العلمية والاسماء التجارية المتاحة وأسعارها فليس كل دواء يناسب كل مريض وكل فئة .

وبالتالي نصيحتي لكل إخواني وأخواتي أن يقرأوا ويبحثوا ويكتبوا ملاحظاتهم وأوراقهم الخاصة في التعامل مع مع حالة معينة .. أذكر أني في بداية الامتياز كنت أحضر أوراقا لي شخصيا وكنت أشرحها لزملائي ليس تكبرا مني ولكن حبا في نقل المعلومة الصحيحة لأن المريض قد يكون يوما من الأيام أحد أقربائي . فلا يجب كتم العلم لأن كاتمه عليه إثم كبير.

فأنا أحب دوما نظام الألجورزم .. بمعني عندما أقابل حالة معينة كيف أتصرف معها تدريجيا .. يعني مثلا عندما تقابل حالة إغماء يجب عليك فعل كذا وكذا.. عندما تقابل حالة ارتفاع درجة الحرارة في الأطفال يجب عليك التفكير في كذا وكذا وتتصرف علي أساس كذا وكذا .. هكذا يجب أن تتعلموا .

وهذا ما قدمته لكم في هذه المذكرات قمت بإضافة ما يقرب من 40 صورة في نهاية هذا الكتاب من الـAlgorithm

ويجب عليكم قراءتها بعناية وقراءة كل موضوع علي حد ومحاولة تطبيقه علي الحالات التي تأتي لكم وستجدون فارق كبير

فستجد نفسك تعرف كيف تتعامل مع الحالة دونا عن غيرك فلا تبخل علي نفسك بأن تحتفظ بالمذكرات معك دوما لترجع إليها باستمرار.

و أحب أن أوضح شيئا .. يجب أن تعرف وأنت طبيب امتياز أن لك سقف في التعامل مع الحالة.. أقصد يكون دورك كذا وكذا ثم يأتي بعدها دور النائب المتخصص أو الطبيب المختص .

مثلا في حالات الذبحة الصدرية أو الاحتشاء القلبي دوري كبداية عندما أري هذه الحالة وضعها علي اكسوجين وعمل رسم قلب لها وإعطائها موسع للشرايين القلبية ومضاد للتجلطات ومسكن للآلام ثم يأتي بعدي دور طبيب القلب.

فهو قابل الكثير والكثير وينبغي أن أتعلم منه كيف يتصرف في هذه الحالات وهكذا وهكذا

لأنها مسئولية عظيمة .. فينبغي عليك أن تعرف ما حدودك في التعامل مع المريض وبعدها يأتي دور الطبيب المختص الذي ستتعلم منه ما بعد ذلك وهناك الكثير من الكتب الجنبية التي أحترمها وينبغي عليكم اقتنائها كما سأوضح لكم بعد ذلك

في النهاية أنصح الجميع بكثرة القراءة والاطلاع ينبغي عليك كل يوم أن تقرأ موضوعا جديد وتعرف حالة جديدة

مثلا في مذكراتي هذه اقرا حالة واحدة يوميا واقرأ موضوعه المدرج كاملا واستخلص منها ما يجب عليك فعله وما تستطيع فعله لأن ما يجب عليك ليس كما تستطيع فعله لأنك لا تدري أي مستشفي أنت فيها وما إمكانياتها ؟ وبعدها تكتب ما ستفعله وتصنع لنفسك ألجورزم خاص بك وتعلمه لأصدقائك.

في النهاية لا أطلب منكم سوي الدعاء لي ولأهلي

1

انت قاعد في أمان الله ويدخل عليك رجل كبير في السن مدخن منذ فترة يشتكي لك من ألم في أعلي البطن epigastrium وحاسس بنهجان Tachyonea وعرقان .

غالبا كله هيعتقد إنها Gastritis عادية وهتديله حاجة AntiAcid وهتعتقد إنه هكويس جدا.

إن فعلت ذلك فقد يموت المريض .

لابد أن تتذكر هذه القاعدة

Epigastric Pain In Old smoker man ..Inferior Miocardial Infarction Until prove otherwise

وبالتالي يجب عليك أن تقوم بعمل رسم قلب سريع

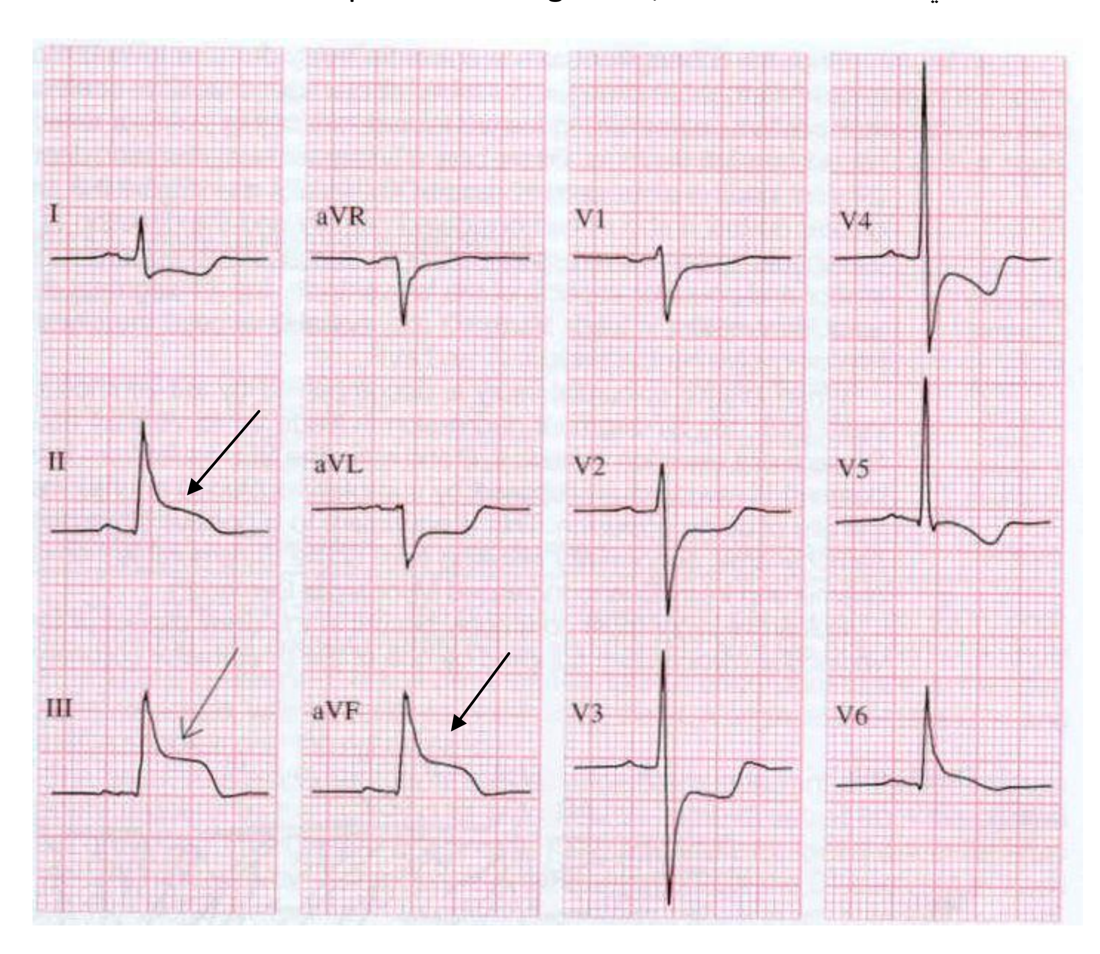
أين ستبحث عن Inferion MI

ستبحث عنها في Lead || , ||| , aVF

كيف تفرق ال Ischemia عن الـ Infarcytion

خلي بالك دوما إنا الـ Infarction معناه ارتفاااع ناقوس الخطر – كلمة ارتفاع يعني raised ST segment

بينما في الـ ischemia هتلاقي Depresses ST segment , Inverted T wave



((ولنا لقاء إن شاء الله عن كيفية قراءة رسم القلب ECG))

ماذا تفعل وأنت طبيب امتياز لهذه الحالة سواء قابلتها في قريتك أو في عيادة أو في استقبال أو اتصال تليفوني حتي:

كما قلنا سابقا طبيب الامتياز له سقف يجب أن يعلمه جيدا ويجب ألا يتخطاه لأننا نتكلم عن حياة مريض

فلو شكيت في الحالة لازم تحولها للمستشفي حالا وانت بتحولها أو وانت في المستشفي ومنتظر نائب الكارديو هتعمل الاتي :

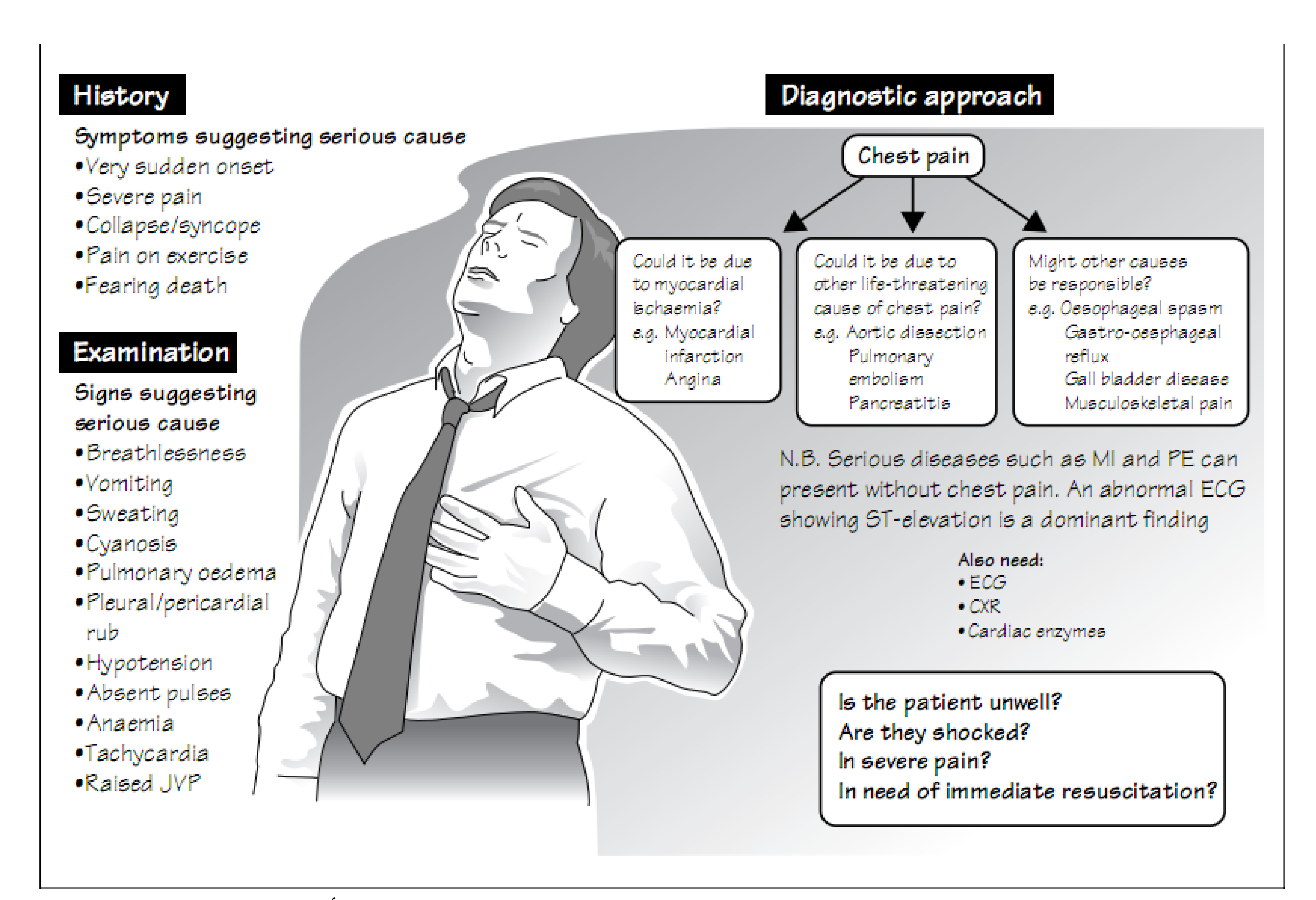
- لابد أن تضع المريض علي أكسوجين في الحال.
- وهو نائم علي السرير هتضع Sublingual Nitrate 5mg

لو أخدها وهو واقف هيقع منك علي الأرض .... as nitrates leads to vasodilatation so, hypotension then collapse

- يمضغ قرصين لـ أربع أقراص أسبرين أطفال 75مجم .
- لاصقة طبية تحتوي علي nitrates

ما تفعله الان يمكن أن ينقذ حياة مريض وبعد ذلك يأتي دور نائب القلب ويمكنك أن تتعلم منه المزيد بعد ذلك

- تذكر جيدا أي حالة فوق ال 50 سنة لابد من عمل رسم قلب إذا أتت لك في الاستقبال.



في الصفحة القادمة مخطط لقراءة رسم القلب وهو يحتاج منك معرفة المعلومات الأساسية في رسم القلب

How to read ECG?

ECG is Either - Rhythm strip - Complete ECG

Rhythm Strip

regular

BradyCardia — **Check QRS shape**

- **Broad and Bizzare :** 3rd AVB
- **Normal :** check P wave
  - Absent : Nodal Rhythm
  - Multiple : fixed 2nd AVB
  - single : sinus bradycardia (It may be 1st AVB so look at P-R interval)

Tachycardi — **Check QRS shape**

- **Broad and Bizzare** : Ventricular tachycardia
- **Normal :** check P wave
  - Absent : supra ventricular tachycardia
  - Multiple : Atrial flutter with fixed block
  - single : sinus tachycardia

Irregular

Check QRS shape

- **Broad and Bizzare :** vent. Extrasystole
- **Narrow , Normal :** check P wave
  - Apsent : AF
  - Multiple: Atrial Flutter with Variable Block Or variable 2nd degree AVB
  - Single : supraventricular extrasystole

Normal

- Atrial flutter with 4:1 block
- 1st AVB

Complete ECG

هنا يجب أن تعلق علي 5 أشياء قبل أن تنطق بالتشخيص وهم كالاتي :

- **Rhythm , rate**
- **Axis**
- **Lead ||** **- V1** **- v6**

Rate = **300/number of small squre between 2QRS**

Axis — **seen in Leads | and AVF**

Rt Axis Deviation : Rt Ventricular ++

Lt Axis Deviation : Lt Ventricular ++

||

Mitrale : Broad & Bifid : LA++

Pulmonale : tall and thin : RA++

Atrial Flutter **Nodal Rhythm**

Absent : AF, Mid Nodal Rhythm

P-R interval : if prolonged : 1st AVB

Infarction : raised ST: In (**||,|||,AVF**) Inferior MI

Ischemia : Depressed ST (**||,|||,AVF**)

V1

Ischemia : depressed ST , Inverted T in (V1,2, 3)

Infarction : Raised ST, (V1 , 2 , 3)

Path. Q : old infarction

RBBB **RV Enlargement (with Rt Axis)**

Ischemia : depressed ST, Inverted T,in (V4,5,6)

Infarction : Raised ST , (V 4,5,6)

Path. Q : old infarction

LBBB **LV Enlargement (with Lt Axis)**

2

استيقظت من نومي في القرية علي نداء لأم كبيرة في السن تتألم بشدة من صداع شديد وألم في العين .
سارعت بقياس ضغط الدم لأجده مرتفع جدا 180\100 بعد ذلك فحصت عينها اليسري لأجد فيها احتقان congestion , fixed semidilated pupil

العين الأخري سليمة. وضغطها مرتفع جدا . حالة طوارئ لابد أن تتصرف .

أعطيتها قرص تحت اللسان لتنزيل الضغط captopril (ACE I) وأخبرتهم بسرعة التوجه للمستشفي لرؤية طبيب عيون لأني أشك في ارتفاع ضغط العين . وأنا علي خبرة بالعلاج الذي يمكن إعطاءه لهذه الحالة . لكن أهل مكة ادري بشعابها كما يقال في المثل . وأنا لست طبيب عيون يمكنهم قياس ضغط العين وإعطاء علاج لتنزيله ثم يمكنهم التدخل في الحالة.

لكن ليتني أعطيت لها العلاج من البداية

سأكمل لكم . ذهبت لأقرب مستشفي مركزي وبعدها كشف عليها طبيب عيون وأخبرها أنها بحاجة للذهاب لمستشفي الرمد في العاصمة . مع العلم إن مستشفيات الرمد كثيرة جدا الخاصة منها . واليوم ليس يوم الجامعة .المهم ذهبت المريضة لمستشفي الرمد وكشف عليها طبيب رمد وأخبرها أن ترجع مرة أخري لطبيب العيون في المستشفي المركزي لأن ضغطها مرتفع ومش هينفع يعملها حاجة إلا لما ضغطها ينزل

للعلم قبل أن تتركني المريضة كتبت لها Frusemide 40 mg IM عائلة الـLoop Diuretics

لتنزيل الضغط المرتفع

المهم لأن الناس غلابة لا تعرف ما يجب فعله في هذه الحالة رجعوا مرة أخري للمستشفي المركزي ، بما يعني ساعة ذهاب وساعة عودة وكل هذا والمريضة تتألم

عندما عادت للمستشفي مرة أخري أعطاها الطبيب العلاج كما يجب أن يكون

R/ Mannitol 20%

R/Acetazolamide (K sparing Diuretic used in Increased Intra Ocular Pressure)

R/Pilocarpind drops

(Miotic drug to drag iris away from eye drainage so, help in draining eye)

هذا هو علاج الطوارئ المطلوب أن تعرفه لمثل هذه الحالات

لكن المأساة أن ذهبت هذه الأم الكريمة للصيدلية ليعطيها دواء مشابة للنقط

وهنا سأكتب أسماء تجارية

القطرة المطلوبة لعمل Miosisاسمها pilocarpine ومن اسمائها التجاريةIsopto-carpine

لكن الصيدلاني أعطاها Isopto-atropine

تشابه في معظم الحروف لكنه تضاد في شغل القطرة . فالصيدلاني أعطاها Atropine

يعني هتعمل severe Mydriasis

يعني هيخلي الـIris يضغط علي drainage of eye and increase IOP

وليست هذه هي المشكلة وحسب المشكلة إن مدة عمل الAtropineهي سبع أيام ولا يوجد مضاد له يوقفه لتظل هذه الأم تعيش هذه المعاناة

أخبرتكم هذه القصة لتعلموا علاج الطوارئ لمثل هذه الحالة ولتنبهوا المرضي علي نوع القطرة

3

كنت في الاستقبال نادتني الممرضة لرجل في الأربعينيات من عمره اشتكي فجأة من احمرار عينه اليمني عند الاستيقاظ من النوم – هو يريد الكشف عند طبيب رمد - . سألته هل رأسه اتخبطت في أي حاجة . فاجاب بالنفي . هل بياخد علاج لضغط أو سكر فأجاب بالنفي . المهم كشف عند طبيب الرمد في نفس الوقت

فأخبره إن عينه سليمة وإنها نزيف بسيط تحت الملتحمة وهيختفي إن شاء الله . المهم قابلت الراجل وقلت لازم أقيسله الضغط ..وبالفعل وجدت ضغطه مرتفع جدا 170 \ 100

أعطيته أمبول frusemide 40 mg وانتظرت مرة أخري لقياس ضغطه والحمد لله نزل وأخبرني إنه أحسن بكتير الحمد لله لأنه كان بيعاني من صداع لكن لم يذكره لأنه كان خايف أكتر علي عينه.

NB: Hypertension is a Silent Killer

لازم تقيس الضغط لأي حالة تيجيلك ومش تستهين بالحالة ولا بشكوتها . ولازم تقيس الضغط صح

الأول : هنتكلم عن كيفية قياس الضغط بطريقة صحيحة بعد ذلك نتكلم عن علاج الضغط نفسه.

The following steps provide an overview of how to take your left arm blood pressure on either a manual or digital blood pressure monitor. Simply reverse the sides to take a blood pressure in your right arm.

1. Locate your pulse

Locate your pulse by lightly pressing your index and middle fingers slightly to the inside center of the bend of your elbow (where the brachial artery is). If you cannot locate your pulse, place the head of the stethoscope (on a manual monitor) or the arm cuff (on a digital monitor) in the same general area.

2. Secure the cuff

Thread the cuff end through the metal loop and slide the cuff onto your arm, making sure that the stethoscope head is over the artery (when using a manual monitor.) The cuff may be marked with an arrow to show the location of the stethoscope head. The lower edge of the cuff should be about 1 inch above the bend of your elbow. Use the fabric fastener to make the cuff snug, but not too tight.

Place the stethoscope in your ears. Tilt the ear pieces slightly forward to get the best sound.

3. Inflate and deflate the cuff If you are using a manual monitor:

- Hold the pressure gauge in your left hand and the bulb in your right.
- Close the airflow valve on the bulb by turning the screw clockwise.
- Inflate the cuff by squeezing the bulb with your right hand. You may hear your pulse in the stethoscope.
- Watch the gauge. Keep inflating the cuff until the gauge reads about 30 points (mm Hg) above your expected systolic pressure. At this point, you should not hear your pulse in the stethoscope.
- Keeping your eyes on the gauge, slowly release the pressure in the cuff by opening the airflow valve counterclockwise. The gauge should fall only 2 to 3 points with each heartbeat. (You may need to practice turning the valve slowly.)
- Listen carefully for the first pulse beat. As soon as you hear it, note the reading on the gauge. This reading is your systolic pressure (the force of the blood against the artery walls as your heart beats).
- Continue to slowly deflate the cuff.
- Listen carefully until the sound disappears. As soon as you can no longer hear your pulse, note the reading on the gauge. This reading is your diastolic pressure (the blood pressure between heartbeats).
- Allow the cuff to completely deflate.

4

استيقظت علي اتصال هاتفي في بلدتنا المتواضعة لأذهب لأري شاب لم يبلغ العشرين من عمره وجدوه في الصباح فاقد الوعي .. لا يحرك ساكنا.. استيقظت وذهبت مباشرة إليهم فإنه نداء الواجب

دخلت علي الشاب لأجد البلدة كلها مجتمعة عنده .. تخيل نفسك تكشف علي مريض وحوله ما يقرب عن ال 30 نفر من البلدة رجالا ونساء أطفالا وشيوخا .. هذه هي قري مصر يقفون جميعا في وقت الأزمات....لكني لا أحب هذه الاجتماعات وقت الكشف .. فعادة آمرهم بالخروج جميعا وليبقي اثنين أو ثلاثة علي الأكثر

المهم ..أهم حاجة إنك لازم تطمن علي

ABC = Airway Breath Circulation

وبنظرة سريعة أجده يتنفس وبه نبض بالطبع لكنه لا يحرك ساكنا

قست له الضغط لأجده مرتفعا .. ولكنه ليس سببا لهذا الفقدان في الوعي

اختبرته عصبيا لأجد أي علامة لـ

Signs of lateralization

لم أجد شيئا ... سمعت قلبه ونفسه لم أجد شيئا

سألتهم هل بياخد علاج لضغط أو لسكر أو لأي مرض

فأجابوا بالنفي . فسألتهم هل أخد أي دواء قبل ذلك .. فأجابوا بالنفي (لكن بعد ذلك علمت إنه تعاطي بعض الأدوية)

ما هذا الموقف الذي لا يحسد عليه أحد ... الناس حولك متجمهرة تنتظر لتري الطبيب الهمام ما يفعل مع المريض

لكني لاحظت أن المريض عرقان وهناك ارتفاع في النبض

tachycardia

NB:pupils are equal and reactive

المهم ..يبدوا أنه ليس المريض وحده الذي يتصبب عرقا الان

آه تذكرت الان معلومة قرأتها في أوكسفورد للطوارئ سأخبرها لكم لاحقا

المهم : أخرجت جهاز السكر وقست له سكر عشوائي لأجده 24 فقط

حمدت الله عز وجل .. وأخبرتهم بسرعة جلب العلاج من الصيدلية المجاورة ..ولكن لحين عودتهم قد يموت المريض فماذا تفعل في هذا الموقف

أخبرت الأهل بعمل ماء بسكر أو هل يوجد عسل أسود أو عسل نحل أو أي شئ مسكر. المهم تم عمل ماء بسكر وأجلست المريض مستندا بظهره علي أحد هؤلاء العدد الغفير ووضعت ملعقة ماء بسكر في فمه .. وسبحان الله

Swallowing reflex is working well

معلومة تذكرتها من شرايط أستاذنا الدكتور أسامة محمود

المهم ما العلاج الذي طلبته من أهل المريض

R/ Glucose 25% or Glucose 10% أيهما متاح

وطبعا لا تنس طلب كانيولا وجهاز محلول وريدي وسرنجة فالدقائق تحدد حياة المريض هنا

وهنا أيضا يجب أن تظهر مهاراتك في تركيب الكانيولا ولكن الجميع ينظرون إليك ..فلا تنس التوكل علي الله

المهم الحمد لله تم تعليق المحلول .. وبعد دقيقة واحدة قام المريض وفاق من غيبوبته ولله الحمد

NB: Becareful if you infuse Glucose 50% it is irritant to veins so give saline in cannula befor and after infusion

NB: Donot infuse less than Glucose 10 %

You can only infuse Glucose 10 - 25 - 50 %

Then recheck blood sugar again

NB: If you infuse 25% infuse 200 ml & if 50 % infuse 100 ml

دعك من كل هذا .. السؤال الان لماذا يدخل شاب لم يبلغ ال20 من عمره في

Hypoglycemic coma?????????????????????

أول سؤال لازم تطرحه عليهم .. هل بيتعالج من سكر ؟ هل بياخد انسولين ؟

إذا كانت الإجابة بالنفي .. فاعلم أن بنسبة كبيرة جدا السبب في هذه الحالة في شاب هو

Drug overdose either insulin or Hypoglycemic drugs

وفعلا كان هذا هو السبب

ألقاكم في حالة أخري

5

كنت جالسا في أمان الله .. لأجد امرأة في العقد الثالث من عمرها تدخل علي لتشتكي عافانا الله وإياكم من ...وجود غشاوة علي عينيها الاثنين .. فبادرت بأن أخبرها بأنها بحاجة لطبيب رمد وليس لطبيب طوارئ لكني تراجعت عن كلامي فقد تكون مريضة ضغط وأنا لا أعلم فآثرت السكوت حتي تنتهي من كلامها .. لتخبرني بأنها ذهبت لطبيب رمد بالفعل ليخبرها بأن عينيها سليمتين الحمد لله . وإنه عليها مراجعة طبيب طوارئ لمتابعة الضغط ..

المهم .. قست لها الضغط لأجده

Low normal 100 / 60

لكنه لن يكون السبب في هذه الغشاوة لأن المريضة أخبرتني أنها تعاني منذ ففترة من انخفاض في الضغط وأنها تعودت علي هذا الانخفاض اللهم إلا من بعض الصداع بين الحين والاخر

حقيقة شعرت بالحيرة .. لأكن أخذت أسألها وآخذ منها تاريخ طبي

سألتها عن أي صدمة

Head trauma

لكنها أجابت بالنفي .. وإن المشكلة منذ يومين .. ولا يوجد تحسن .. أخذت أسألها عن أي مرض مزمن .. فأجابت بالنفي .. سألتها هل أصبتي بدور برد من أسبوع .. فهزت رأسها بالموافقة .. لكنها لم تتناول أي علاج له .. فسألتها عن صداع أو تقل في رأسها يزداد في الصباح .. فأجابت بنعم .. وإن الصداع يزداد مع السجود في الصلاة

سألتها عن رشح من الأنف ..فأجابت إنه بسيط

المهم .. شعرت بالحيرة لأن وجود غشاوة علي عينيها ليس بالأمر الهين فماذا أفعل ؟

قررت أن أعطيها علاجا للضغط المنخفض .. وكل أسئلتي التي ذكرتها .. تدل علي شئ واحد تقريبا وهو أنها كانت تعاني من دور برد من أسبوع وحدث لها مضاعفات بسببه وانقلب إلي

sinusitis

وهو أمر يجب أن يعلمه طالب الطب فضلا عن طبيب الامتياز

فصارحتها بالأمر وقررت إعطائها علاجا لالتهابات الجيوب الأنفية

لكن بشروط .. االعلاج يجب أن يستمر لمدة أسبوعين متتاليين

وأعطيتها علاجا للضغط المنخفض مع بعض المسكنات

وذهبت المريضة

أتت المريضة لي مرة أخري بعد أسبوع .. لأجدها تخبرني أن الحالة تحسنت الحمد لله شبه نهائيا .. ففرحت بشدة لأن الله وفقني للتشخيص وأن المريضة أصبحت تري من جديد بلا أي معوقات.

ما العلاج إذا؟؟؟؟ لن أذكر أسماء تجارية

R/ Quinolones

R/ Diclofenac sodium

R/ Nasal decongestant or Systemic decongestant

To open sinuses مع بعض التعليمات مثل : الاكثار من شرب الماء لأن الماء

Water is the best Mucolytic

واستنشاق بخار الماء لفتح مسام الجيوب الأنفية

6

كنت جالسا في الاستقبال أتصفح كتابا طبيا .. لتدخل علي مريضة .. تشتكي من هرش شديد جدااااا في كل جسدها .. ومع الهرش احمرار شديد في الجسم ..وتشعر بدوخة .. ليس من البديهي الان أن تسأل عن سبب الهرش

لأن المريضة قد تدخل منك في صدمة الان

Anaphylactic shock

لماذا يا شطار؟؟؟

ليس الان .. لأن المريضة قد تموت منك فلا تستهين بهذه الحالة لأن المريض يأتي محرجا إنه بيهرش

يجب أن تعطيها الان حقنة لمضادات الهيستامين

Antihistaminics

وبعدها تجد نتيجة سريعة إن شاء الله .. ويمكنك تكرارها مرة أخري

فماذا لو لم تستجيب يجب أن يكون لك دوما خطة بديلة .. فتعطيها

Systemic Steroid

لكن قبلهم يجب أن تضعها علي أكسوجين لأنه قد يحدث لها

Bronchospasm

فماذا لو لم تستجب لما سبق

يجب عليك أن تعطيها أدرينالين ..ولكن كيف نحسب الجرعة وكيف نعطيها؟؟؟؟

هنا استدعيت نائب العناية ليقوم بذلك ..لا تنسوا أن الأدرينالين

Epinephrine is a Strong Sympathomimetic and could kill the patient

وقد تحتاج المريضة لمتابعة النبض ورسم للقلب ووو

لنكتب روشتة للعلاج إذا

وبلا أسماء تجارية

R/ Diphenhydramine 25 – 50 mg IV q6hr

R/ Hydrocortisone 300 mg IV bolus

نأتي للكلام المهم وهو كيف نعطيها الأدرينالين؟؟؟؟؟

في الكتب ستجد مكتوب بالنص

- Administer 0.3 – 0.5 ml of 1:1000solution subcutaneously …. And could repeat after 20 minutes if necessary

معناها إنك هتحل 1ملج من الأدرينالين علي 1000 سم من محلول وهو صعب

وبالتالي هتحل نصف ملجم من الأدرينالين علي 500 سم من المحلول وتكون حصلت علي التركيز المطلوب

وهنا يأتي السؤال لو المريضة دخلت في صدمة

Anaphylactic shock

هل هينفع أديه تحت الجلد؟؟؟؟؟ أكيد لا !!!!! فما الحل إذا ؟؟؟

نفس الجرعة لكن ستعطيها

Sublingual

ولكن للأسف هذه المريضة لم تستجب حتي الان لكل هذا الجرعات وقد حدث بالفعل

فماذا يجب أن نفعل؟؟؟؟؟؟؟؟؟؟؟؟؟؟؟؟؟

حاليا أسألها عن سبب الحساسية .. هل أخذت أي دواء ؟؟؟

هل اتقرصت من أي شئ؟؟ هل أكلت أي نوع من أنواع الطعام ؟؟

والمريضة تجيب بالنفي للأسف

ومازالت تعاني من الهرش الشديد؟؟؟

فما الحل إذا؟؟؟

In Books you will find this?

Add 1mg of epinephrine to 500 ml Dextrose and infuse it by 1ml per minute

وتبحث عن السبب لأنه إذا عرف السبب بطل العجب وأهم علاج إنك

Stop The Offending antigen

7

جاءت الأم مسرعة حاملة طفلها الذي لم يبلغ الشهر السادس من عمره.. وتقول الحقني يا دكتور بيصرخ ويعيط بدون أي سبب.. آآآآآآآآه لا أحب الطوارئ .. تسأل الأم حصل إيه هل وقع هل اتخبط .. فتجيب بالنفي ؟؟

هل أكلتيه أي شئ جديد يا ماما .. آه يادكتور بياكل كل حاجة في البيت.

وضعت اصبعي في فم الطفل .. فلم أجد suckling إذا ليس هو الجوع فالطفل يبكي من الجوع كثيرا .

بدأت أسترجع أشهر سبب للبكاء في الأطفال بدون سبب وهو Abdominal Colic

فبدأت بعمل ما يسمي بتمارين المغص .. وهي أن تجعل الطفل علي ظهره .. وتمسك بكلتا قدميه وتبدأ في تحريك رجليه علي بطنه وترجعهم مرة أخري ..وكررت هذه التمارين .. وبدأ صراخ الطفل بالهدوء.

آآآه الحمد لله إنه المغص ..

وبعدها علمت الأم كيف تعمل هذه التمارين وأعطيتها دواء للمغص . وأهم نصيحة هي تكريع الطفل وسط الرضاعة وبعد الرضاعة لطرد الغزات من المعدة

والسؤال هنا : ما أسباب البكاء عند الأطفال ؟ سأسرد الأسباب الرئيسية وسيتم الشرح لاحقا إن شاء الله

- الجوع - الحاجة للنوم - الحاجة للحمل بواسطة الأم أو الأب واللعب - المغص - التهابات في الحفاظات - عمل حمام _أعزكم الله_
- حاجة الطفل للتكريع خصوصا بعد الرضاعة - المرض

R/ Simethicone drops

15 نقطة بالفم 3 مرات يوميا

8

دائما هناك مريض بعد الفجر .. ستجرب هذا في كل نبطشياتك فلا تعجب
فزوار الفجر حاليا ((قديما كانوا امن الدولة)) كثر أشهرهم مريض الذبحة الصدرية والأزمة الصدرية ..
وهذا المريض الذي نحكي عنه.
يدخل المريض وهو يمشي بصعوبة واضعا يده علي أحد جانبي بطنه ويقولك مغص شديد جدا يا دكتور .
بعد أن ينام للفحص ويككشف بطنه تسأل هل ده وجع ثابت ولا مغص بيروح ويجي .. فيجيبني بيروح ويجي يا دكتور ده خلاني أرجع من شدة الألم
فتبادره بسؤال آخر.. هل الألم ده بيسمع في الخصية .. فينظر متعجبا آه يا دكتور عرفت ازاي .. فبعد أن يتملكك الشعور بالفخر للحظات .. تسأله حدد لي مكان الألم فيضع يده علي مكان هو تشريحيا مكان الحالب ureter
فتأخذ نفسا عميقا وتتنهد عندك مغص كلوي يا أستاذ
هل فيه دم في البول .. قد يجيبك بنعم أو بلا والصندوق يحدد في النهاية . احم احم .. أقصد تحليل البول سيحدد فقد يوجد دم لكنه غير مرئي microscopic hematuria
لكن لحظة .. يجب أن أكمل الفحص لأستبعد أي سبب آخر فمن شدة الألم قد يختفي معه عارض آخر...لا تنس أن تستبعد الزائدة الدودية فما اكثرها ..ففي حالة شبيهة كان مع المغص الكلوي في الجانب الأيسر من البطن ظهر في اليوم الثاني ألم بالجانب الأيمن من البطن .. وبدأ الإحساس بعدم الرغبة في الأكل nausea وبدأت الحرارة في الارتفاع .. وبدأ الألم لا يستجيب لأي علاج .. فلا تنس أن تستبعد الزائدة الدودية فقد تنقذ مريضا من الموت
بعد أن استبعدت الالتهابات بالزائدة الدودية ... يجب أن تعطي المريض العلاج لكي يستريح من هذا الألم الشديد وهو يرضي بألم لكن أقل حدة.
والعلاج يسير والشفاء بيد الله عز وجل

R/ NSAID injection better to use IV route

R/ Spasmolytics like Buscopan

R/Calcium channel blockers are smooth-muscle relaxants. In combination with prednisolone, they have facilitated ureteral stone passage in several small prospective studies.

Nifedipine facilitates the passage of ureteral stones. The extended-release formulation simplifies treatment and encourages compliance. Only short-term therapy (10 d) should be considered for this indication.

لكن السؤال الذي يطرح نفسه ماذا ستفعل مع المريض بعد أن يهدأ الألم؟؟؟؟؟؟؟؟؟؟؟؟؟؟

هل ستحوله علي عيادة المسالك ليتابع حالته؟؟؟؟ أم أنك سترشده للطريق الصحيح وبعدها يحول للمسالك؟

يجب أن تعرف سبب هذا المغص ويجب أن تعالج السبب . فيجب عليك طلب منه تحليل بول لمعرفة نوع الأملاح . ولمرفة هل يوجد صديد أم ماذا وبناء عليه سيأخذ العلاج الصحيح
يجب طلب أشعة تلفزيونية لإظهار أي حصوات أو لإظهار أي اسباب أخري فقد يوجد

Appendicular mass

كما وجدت مرة في حالة ولكنها ظهرت بعد المغص الكلوي بيوم. والمريض بياخذ مسكنات وبالتالي بيعمل

Masking to Pain so Appendicitis may be Misdiagnosed

وبناء علي نتائج التحاليل والأشعة سيتم تحديد العلاج.

ملحوظة : هناك تعليمات يجب أن تعطيها للمريض قبل خروجه من الطوارئ مثل : الإكثار من شرب الماء .. الابتعاد عن أي أطعة بها نسب أملاح مرتفعة

وإلي لقاء في حالة أخري

Common Causes of Abdominal Pain

abdominal pain	Frequency	Common symptoms	Common signs	Important investigations
Appendicitis	Common	Central abdominal pain, then localizing to right iliac fossa Fever, anorexia	Right iliac fossa tenderness, rebound, guarding	
Infective gastroenteritis	Very common	Vomiting, diarrhoea, diffuse abdominal pain	Dehydration Diffuse abdominal tenderness	Stool culture
Peptic ulcer	Very common	Epigastric pain Can radiate to back Increased certain foods Alleviated antacids	Epigastric tenderness Acute abdomen if perforation	Upper GI endoscopy
Oesophageal reflux	Very common	Burning retrosternal, epigastric pain Alleviated antacids Exacerbated at night, lying flat	None	Upper GI endoscopy
Biliary colic	Rare	Sudden onset, severe right upper quadrant pain May have vomiting	Right upper quadrant tenderness	Abdominal ultrasound
Cholecystitis	Common	Right upper quadrant or epigastric pain Exacerbation with fatty foods	Right upper quadrant tenderness Fever	Abdominal ultrasound
Pancreatitis	Common	Severe, epigastric pain Can radiate to back Vomiting	Epigastric tenderness Signs of acute abdomen May have shock, breathlessness	Amylase Abdominal CT
Bowel obstruction	Common	Vomiting Absolute constipation Abdominal pain	Abdominal distension Generalized tenderness Tinkling bowel sounds	Abdominal X-ray
Diverticulitis	Common	Pain especially left lower quadrant Fever Change in bowel habit	Fever Tenderness Acute abdomen if perforation	Abdominal CT
Aortic aneurysm	Common	Central abdominal pain Back pain Sudden and severe with ruptured aneurysm	Expansile, pulsatile mass Shock with ruptured aneurysm	Abdominal CT Abdominal ultrasound
Renal colic	Common	Sudden onset, severe pain in loin radiating to groin or testis Pain may wax and wane Haematuria	Loin tenderness Dipstick urine positive for blood	Plain KUB X-ray IVP Ultrasound

9

دخل علي زوج المرأة المسكينة التي تعدت الخمسين من عمرها ليخبرني سرا أنها مصابة بسرطان علي المخ ، وهي الان تشتكي فقط من انتفاخات.. فأدخلتها لأسألها خير يا أمي ..قالت أنا بطني منفوخة بقالي 4أيام .. طيب يا أمي هل بتعملي حمام براز .. وهل فيه خروج ريح ..((لازم تسأل المريض أسئلة مباشرة ..ولا حرج في ذلك)) فأخبرتني أنها لم تدخل الحمام من يومين ولم يخرج منها أي ريح.. فسألتها هل فيه ترجيع يا أمي ..فأجابت بالنفي .. فسألتها هل فيه ألم في البطن فأجابت بنعم.. فقمت لأكشف علي بطنها لأجدها منتفخة جدا وظاهر عليها

Visible intestinal loops

وسمعت بالسماعة لأسمع أصوات شديدة

Audible intestinal sounds

المهم ..هذه الحالة تعتبر

Surgical emergency

كطبيب امتياز يجب أن تفعل شيئين في هذه الحالة .. أولا عمل إنيميا أو حقنة شرجية ثانيا تركيب كانيولا وإعطاء المريض محلول خوفا من الدخول في

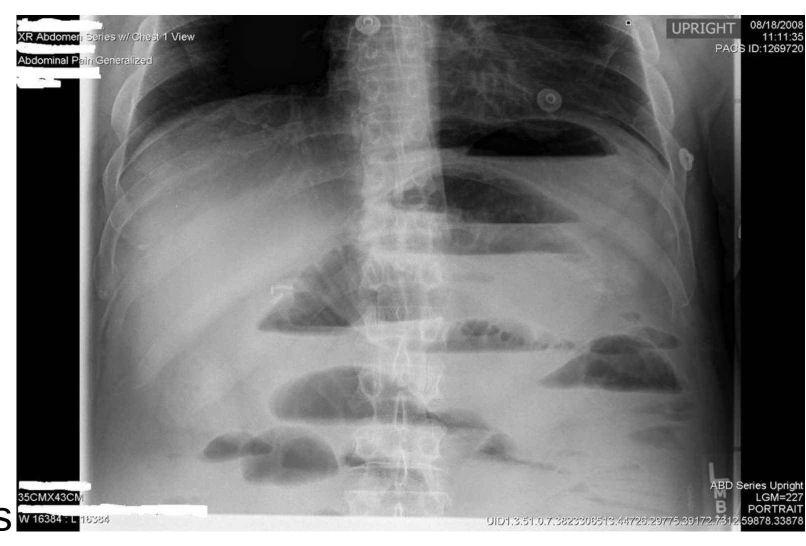
Dehydration

ويمنع تناول أي أطعمة أو مشروبات ، فطلبت من زوجها ذلك عمل حقنة شرجية وطلبت تركيب محلول وريدي وعمل أشعة عادية علي البطن والمريض واقف ، وأشعة تلفزيونية

Abdominal X ray erect position and Ultrasonography

بعدها بساعة اتصل بي ليخبرني أن الحقنة الشرجية الحمد لله ساعدت معها

فأخبرته بضرورة تركيب المحلول الوريدي وضرورة عمل الأشعات المطلوبة



Notice : multiple Air fluid Levels

10

لا يمكنك أن تنسي منظر هذا الطفل الصغير.. عمره لا يتجاوز العاشرة .. يدخل عليك في الاستقبال محمولا علي يد أهله .. نظرته هنا وهناك يبدوا تائها.. تجده فاتحا لفمه ويتنفس بسرعة وتجد فمه جافا جداااااا .. ويضع يده علي بطنه من الألم ..هذا المنظر الشهير لحالة الـ

Diabetic Ketoacidosis

فتسأل أهله بسرعه بياخد علاج لأي حاجة .. فيجيبوك آه يا دكتور عنده سكر وبياخد انسولين

فما السبب إذا ؟؟؟ أكل دون أن يأخذ الجرعة .. فارتفع السكر بشدة عنده ويكون عنده أجسام كيتونية .. أو عاني من أي مرض شديد الفترة الماضية وكان بحاجة لزيادة جرعة الأنسولين .

هذا الطفل سيدخل في جفاف إن لم يكن دخل أصلا ...يجب تركيب كانيولا بسرعة له وتركيب محلول ملح حتي لا يموت من الجفاف.

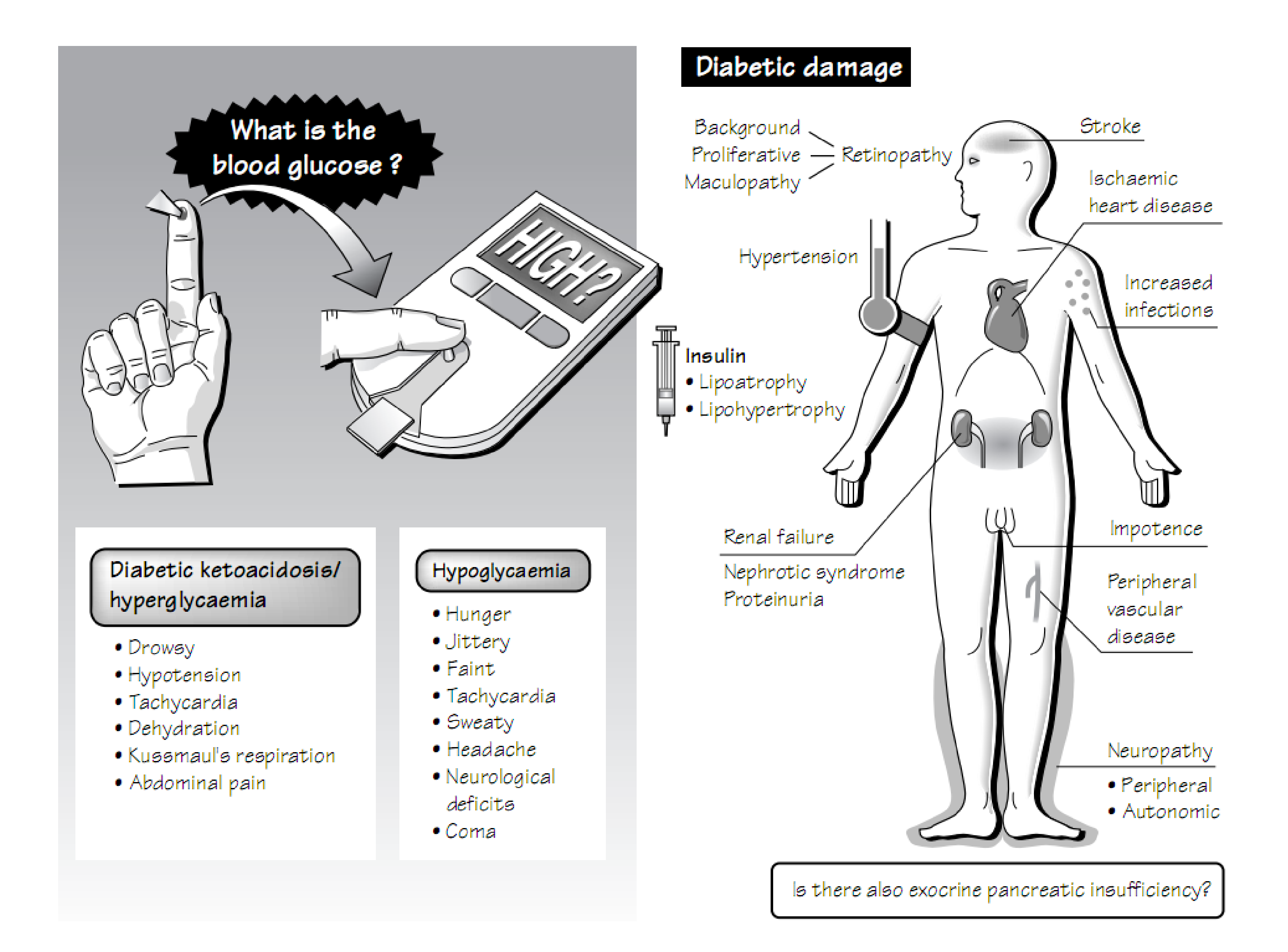
تقيس له السكر تجده يفوق ال 500 تقيس له الضغط تجده منخفضا تقيس نسبة الأجسام الكيتونية في البول تجدها موجودة .

هذا الطفل يجب دخول العناية المركزة للاهتمام بأمور لابد أن تكون في ذهن كل طبيب

Managing diabetic ketoacidosis (DKA) in an intensive care unit during the first 24-48 hours always is advisable. When treating patients with DKA, the following points must be considered and closely monitored:

- Correction of fluid loss with intravenous fluids
- Correction of hyperglycemia with insulin
- Correction of electrolyte disturbances, particularly potassium loss
- Correction of acid-base balance
- Treatment of concurrent infection, if present

لذلك تقوم بتركيب كانيولا بسرعة جدا وتضع له محلول ملح بسرعة مع خمس وحدات انسولين مائي في الوريد مباشرة وتقوم باستدعاء نائب العناية بسرعة.


What is the blood glucose ?
HIGH?
Diabetic ketoacidosis/ hyperglycaemia
• Drowsy
• Hypotension
• Tachycardia
• Dehydration
• Kussmaul's respiration
• Abdominal pain
Hypoglycaemia
• Hunger
• Jittery
• Faint
• Tachycardia
• Sweaty
• Headache
• Neurological deficits
• Coma
Diabetic damage
Background
Proliferative
Maculopathy
Retinopathy
Stroke
Ischaemic heart disease
Hypertension
Increased infections
Insulin
• Lipoatrophy
• Lipohypertrophy
Renal failure
Nephrotic syndrome
Proteinuria
Impotence
Peripheral vascular disease
Neuropathy
• Peripheral
• Autonomic
Is there also exocrine pancreatic insufficiency?

11

قاعد في أمان الله ونفسك تحدثك .. الحمد لله نبطشية هاااااديييييييية ... وقبل أن تكمل حديث النفس تخبرك الممرضة دكتور في حالة جاية وشكلها تعبان جداااااا ... وأنا أراجعها انتي متأكدة

انتي متأكدة ... وتدخل الحالة ومعها اهلها ... إنها نفس البنت التي رأيتها مسبقا أهلها يريدون لها جلسة تنفس بسرعة .... وأنا أسألهم خير يا ماما حصل إيه تاني..

قالوا : كانت بتغسل يا دكتور واستعملت فينيك و كلور و فلاش كلهم علي بعض فطلع منهم روائح غريبة ونفاجأ إنها بتكح جامد ومش قادر تاخد نفسها.... بسرعة جداااااااا سمعت صدرها لقيته

Inspiratory and expiratory wheeze

وأنا أسمعه أخبرت الممرضة أن تجهز جلسة التنفس النيبوليزر 2سم محلول ملح + 1سم فاركولين

وفي نفس الوقت الممرضة الأخري أخبرتها بتجهيز كانيولا بسرعة... وفي أقل من 15 ثانية ولله الحمد قمت بتركيب الكانيولا في نفس الوقت التي تضع لها الممرضة قناع النيبوليزر

وأعطيتها في الكانيولا حقنة مضادة للحساسية ، وأخبرت الممرضة بتجهيز حقنة هيدروكورتيزون احتياطي.

بعد دقيقة سمعت صدرها مرة أخري وجدت أن الحالة تحسنت إلي

Only expiratory wheeze

فحمدت الله وأكملت الجلسة لأجد في النهاية أنه بقي تزييق خفيف جداااااااا

وإن الحالة تحسنت الحمد لله

وبعدها جلست مع الأهل وأخبرتهم أن العلاج للحالة ليس بالجلسات بل في التخلص من كل هذه المواد المطهرة من المنزل بصفة عامة واستعمال بدائل أخري

وعدم التعرض لأي مواد تسبب حساسية للصدر

وأعطيتهم موسع للشعب الهوائية علي هيئة

Salbutamol

بختين كل 6 ساعات لحين تحسن الحالة ثم تعرض علي الطبيب مرة أخري

لذلك : يجب علي كل طبيب امتياز ألا يترفع عن تعلم كيفية تركيب الكانيولا وبسرررررررررررعة وليس عيبا أن تتعلمها من ممرضة أو من أي أحد المهم أن تركبها بسرعة في النهاية فأنت تنقذ حياة مرضي

تصبحوا علي خير

إن بي : لا تنس دوما في مريض الربو الشعبي أن تفحص القلب وتسمعه جيدا وأن تبحث عن علامات الـ

signs of right side heart failure

اللى هى

lower limb oedema,congested neck veins,enlarrged tender liver

لماذا؟؟

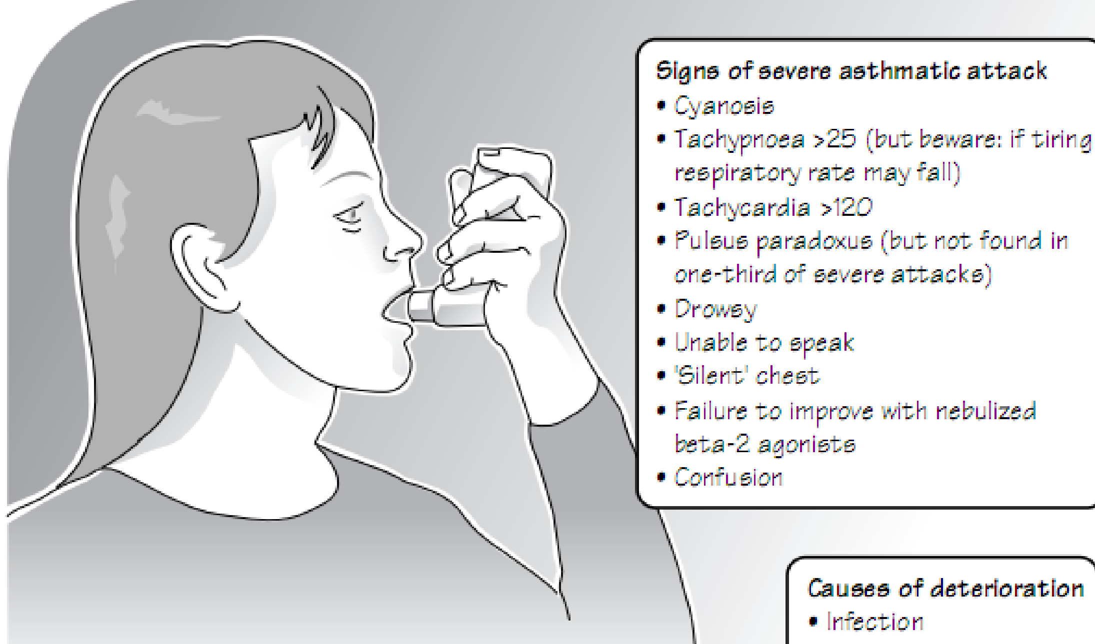
لأن ال long standing B.A بتعمل PULMONARY HYPERTENSION اللى بيؤدى اللى right side heart failure

History

- Wheeze
- Breathlessness
- Chest tightness
- Cough

Examination

- Well/unwell ?
- Tired
- Able to talk in complete sentences ?
- Respiratory rate
- Pulse rate
- Pulsus paradoxus
- Use of accessory muscles
- Intercostal recession
- Wheeze



Severe

- Cyanosed
- Drowsy
- Pulse 130
- RR 35
- Pulsus paradoxus 40 mmHg
- Can't speak

Mild

- Pink
- Alert
- Pulse 80
- RR 15
- No pulsus paradoxus
- Speaking in sentences

12

تلقيت اتصالا هاتفيا من أحد أقربائي ليخبرني أن أحضر له بسرعة .. وكان الوقت تقريبا الثانية بعد منتصف الليل .. والجو شديد البرودة .. وما أحلي النوم في البرد .. لكنه نداء الواجب .. ونداء القرابة اللذان لا تستطيع الهروب منهما .

المهم ذهبت لأجد أن ابنتهم مصابة بدوخة شديدة مع صداع .. ويوجد نهجان شديد ..ويوجد إحساس بالقئ الشديد .. مع القئ ..وتشعر البنت بسرعة ضربات قلبها..فقمت بإجراء الكشف الطبي عليها لأجد أن نبضها متسارع وضغطها منخفض ويوجد سرعة بالتنفس .

بنظرة سريعة حولي وجدت أنه يستعملون للتدفئة في الشتاء ((الفحم))

يسخنوا الفحم علي النار وبعد كده يستعملوه في أطباق كبيرة للتدفئة. والمكان تقريبا محكم الغلق

فتذكرت شيئا درسناه في الفرقة الرابعة وهو التسمم بأول أكسد الكربون

Carbon monoxide poisoning

نتيجة الاحتراق الغير كامل للفحم أو للخشب.

قمت بفتح جميع الأبواب والنوافذ في هذا المنزل و أمرتهم بالتوجه للهواء الطلق وأخذ نفس عميييييييييق جداااااااااا

هذا هو ما أستطيع تقديمه فلا توجد وسيلة للنقل الان للمستشفي والوقت متأخر والبرودة شديدة

ولم أنس إخراج الفحم خارج المنزل تمامااااااااا

في غضون الساعة كانت الحالة قد تحسنت والحمد لله

عندما تأتي هذه الحالة للمستشفيات يجب وضعها علي أكسوجين بسرعة وبتركيز مرتفع

لأن أول أكسد الكربون يتحد مع الهيموجلوبين ليكون

Hb-Co

والذي لا يتحد مع الأكسوجين بعدها وبالتالي لا يصل الأكسوجين للدماغ للتغذية

آآآآه تذكرت أن عائلة بالكامل قد ماتت بسبب هذا الاختناق من قبل وماتت أيضا الحيوانات التي كانت بالمنزل .

يجب أن يوجد بديل لهذا الفحم في التدفئة . وأبسط بديل الملابس الثقيلة.

ودمتم سالمين جميعا

ولا تنسوا التنويه علي الناس مع دخول الشتاء علي هذا الأمر لأنه يحدث معي في كل شتاء

في وقت كتابة هذه القصة كان قد كلمني أحد الأشخاص في المسجد عن نفس الحالة لأبنه في نفس الوقت من الليل وبنفس الأعراض.

إذا إنه الفحم يعلن الحرب علينا فيجب أن نستعد له .

13

ما أشد برودة هذا اليوم .. أنت تمني نفسك بالنوم تحت البطانية وتستمع بالدفء ومشاهدة التلفاز

لكن دائما يوجد من يعكر هذا المزاج .. لكنه نداء الواجب .. يجعلك تشعر بالسعادة عندما ييسر الله لك تشخيص الداء وشفاء المريض علي يدك بإذن الله عز وجل

تدخل عليك الأم ممسكة بابنتها التي تعدت الرابعة من عمرها بقليل .. وهذه البنت الجميلة تبكي ويدها علي أذنها باستمرار .. تخبرك الأم إن حرارتها مرتفعة وايديها علي طول علي أذنها وبتهرش فيها... فسألتها هل كان عندها دور برد من أسبوع يا ماما علي هيئة رشح وسخونية خفيفة ... فتجيبك بنعم

قمت بالكشف علي الطفلة وقست حرارتها لأجدها تعدت ال 38.5 وكشفت علي حلقها لأجد احتقانا بسيطا ... كشفت علي أذنها لأجد آثارا للهرش .. للأمانة لا أحد يحمل منظارا للأذن

Otoscope

فهو غال الثمن .. وكثير من أطباء الأطفال لا يستعملونه إلا من رحم ربي.. فهم يقولون أن التاريخ المرضي كاف.... ارتفاع الحرارة خصوصا بعد دور برد مع وجود ألم بالأذن كاف لتشخيص هذه الحالة خصوصا إذا كان الطفل يستيقظ بالليل ليبكي دون سبب معروف وقد يدخل الطفل في تشنجات بسبب ارتفاع الحرارة في هذه الحالة

إنها حالة اللالتهابات بالأذن الوسطي

ولو أكرمك الله وشتريت منظار الأذن ولو أكرمك الله وقدرت تدخله فيأذن طفل دون أن يتلوي منك ويصرخ فتتمني أن تنتهي بسرعة حتي لا تسمع هذا الصراخ الذي يجعلك قد تفقد أعصابك لكن هيهات فالأهل موجودون ينتظرون الطبيب

إن نظرت في الأذن من الداخل ستجد أن الطبلة فيها احمرار شديد ولا يوجد أثر للضوء من الكشاف

بالعربي :

Congestion and bulging of ear drum & loss of cone of light

في هذه الحالة ستكتب الروشتة المعتادة

Antibiotic combination like amoxicillin + clavulinic acid

Analgesic and antipyretic

ويجب أن تعرف أنها ممنوعة قبل سنتين Local nasal decongestant

وعندما تستعملها تستعمل مرتين يوميا لمدة 3 أيام ولا تكرر....ودورها هو

Shrinkage of Eustachean tube opening to help draining

أهم شئ يا زملائي الأعزاء في الأطفال هو أن تتعلم كيف تحسب جرعة المضاد الحيوي

وتحسب الجرعة علي حسب وزن الطفل ... وعلي حسب الحالة هل هي شديدة أم بسيطة أم متوسطة .. وأيضا جرعة المسكنات يجب أن تتعلم كيف تحسبها

ولنتكلم عن طريقة حساب الجرعة

14

وأنت جالس في النبطشية بتلعب بلاي ستيشن مع زمايلك ((لقتل وقت الفراغ في النبطشية خصوصا في الشتاء)) إذ تسمع صراخا من الخارج فتقوم قبل ما يدخل في جووون وتعمل

Pause

وتقول أشوف مين اللي تعبان ... لتخرج فتجد فتاة في العشرين من عمرها تنحني بظهرها للأمام قليلا وتضع يدها علي جنبها اليمين وتصرخ من الألم ... فتجعلها تنام علي السرير وتسأل أهلها خير يا جماعة .. تخبرك أمها يا دكتور تعبانة من بالليل اليوم اللي فات وبطنها واجعاها جامد ... فتسألها ليه يا ماما مش كشفتوا علي طول .. تقولك الحجة الطبيعية للمريض المصري أصلنا قلنا ده دور برد في المعدة وهتخف .. لكن الألم بيزيد .. المهم عودت نفسي ألا أجهدها في كثرة السؤال .. لكن ما يزيدك غيظا إن مريض يجيلك الساعة 3 الفجر بابنه سخن ..فتسأله هو سخن من امتي.. يقولك من 3 أيام .. طب ليه مش جبته من 3 أيام.... وليه مش بتيجي بدري ليه .. فلا تجد جوابا مفيدا .. المهم نرجع للبنت المريضة

سألتها هل هي مدام ولا آنسة .. فتخبرني بإنها آنسة .. ((ملحوظة تسأل لتستبعد إنه يكون حمل خارج الرحم)) فتسأل آخر دورة كانت متي؟؟؟؟؟ فتجيبني بإنها كانت من 4 أيام فتطمئن إنها ليست حمل خارج الرحم وإنها مش مغص الدورة

المهم سألتها الوجع بدأ ازاي يا ماما .. فأخبرتني إنه بدأ حول السرة .. وبعد كده اتنقل لجنبها اليمين ... فسألتها أخبارك مع الأكل إيه .. فأجابت إن نفسها مسدودة عن الأكل من الصبح

وفيه غممان نفس جامد ...وإنه حصلها اسهال مرة فتسأل هل فيه دم في البراز فتجيبك بالنفي . وأنت بذلك تنفي التهابات بكتيرية بالجهاز الهضمي ... فتسأل وانت تشك في التهابات الزائدة الدودية .. هل فيه سخونية ... فتجيبك بنعم ...

فقمت بالكشف عليها لأجد الضغط منخفض بعض الشئ مع ارتفاع في ضربات القلب وارتفاع في درجة الحرارة

فكشفت علي بطنها لأجد الألم شديد جدا في الجانب الأيمن من الحوض

بالعربي يعني

Maximal tenderness at McBurney's point

فتضع يدك علي هذه النقطة وتسأل المريضة إنها تكح ..((تسعل يعني)) فأجابتني إنها غير قادرة علي ذلك ... فتقوم بعمل

Palpation at this point

لتجدها تصرخ من الألم

إذا إنه

Rebound tenderness

يبدوا إن شكي في محله .. لكن هيهات لا نتقوف عند هذه النقطة .. إنها امرأة يجب أن تطمئن عليها

Gynecologically

فطلبت منهم عمل أشعة تلفزيونية علي الحوض والبطن ..((وأنا أحدث نفسي أن أبحث عن أي مشاكل في المبايض كــ

Ovarian cyst , ectopic pregnancy ,ureteric calculi

وتطلب أيضا تحليل بول وتطلب صورة دم كاملة ((بحثا عن ارتفاع في كرات الدم البيضاء))

تذهب المريضة بعد أن أخبرتها إنه ممنوع أي طعام أو شراب وبعد أن علقت لها محلولا مضاف عليه مضاد حيوي ومضاد للتقلصات .. ولم أضع مسكنا للآلام الان فليس وقته .

بعدها بنصف الساعة تأتيني بالأشعة التلفزيونية ..لأجد

Appendicular mass at McBurney's point

إذا إنها التهابات الزائدة الدودية ... ولم أنس الاطلاع علي صورة الدم لأجد

Lecucytosis with shift to left ((mean predominance of PMN cells))

عاجلا استدعيت نائب الجراحة ليقوم بالكشف علي الحالة .. ليؤكد التشخيص وليجهز المريضة لعملية استئصال الزائدة الدودية .

آآآه الحمد لله أن وفقني لإنقاذ روح انسان .. لو حدث أي تأخر في التشخيص قد يحدث انفجاؤ لهذه الزائدة ولهذه مشاكل كثيرة جداااااااااااا

المهم لازم تلحق وتشوف دورك في البلايستيشن وصل لفين.

نلقاكم في حالة أخري

إن بي : لما تيجي تقرأ في الكتب المحترمة عن أسباب آلام البطن الحادة ستجدهم يقسمونها ل،

Surgical and medical causes

ينبغي عليك حفظهم جميعا .. وننوه عن حالتين مهمين جدا

-DKA

-Inferior MI

يأتون بآلام حادة في البطن غالبا.

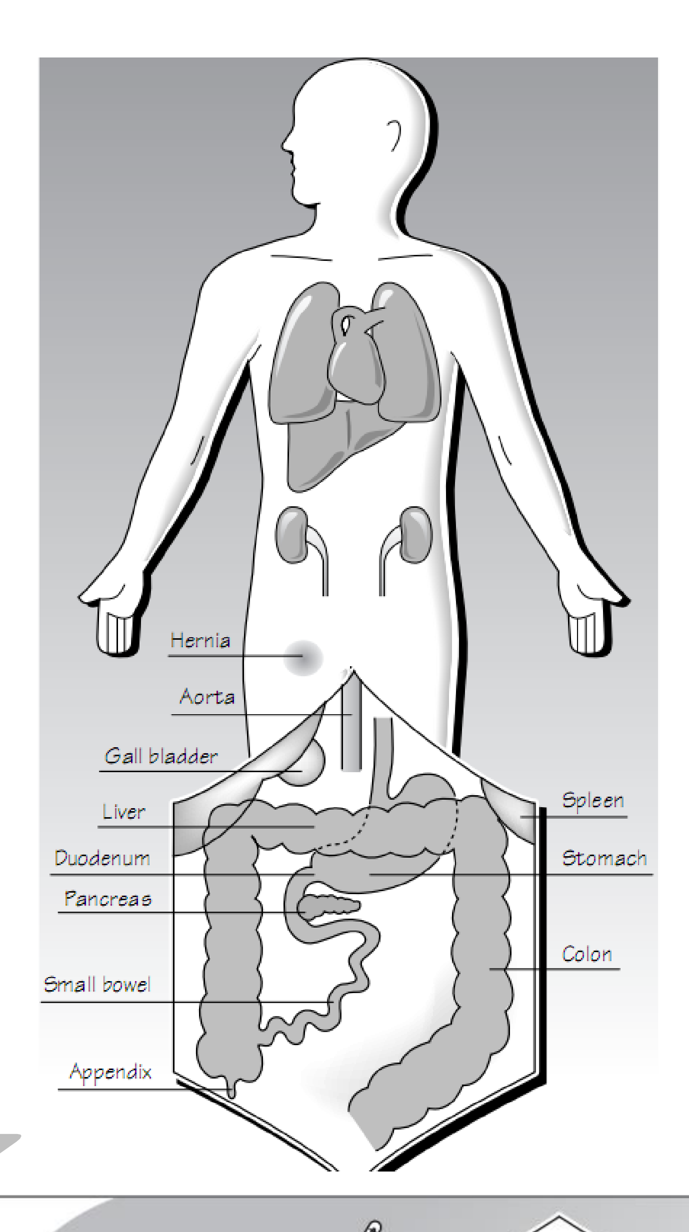
History

Where is the pain?
What type?
Radiation
Precipitation
Alleviation

Other symptoms

Medication

Remember
Extra-abdominal sources of pain, e.g. myocardial infarction, metabolic disturbance



Examination

Well/unwell
Systemic signs
- fever
- shock

Acute abdomen
- tenderness
- rebound
- guarding
- absent bowel sounds

Rectal examination

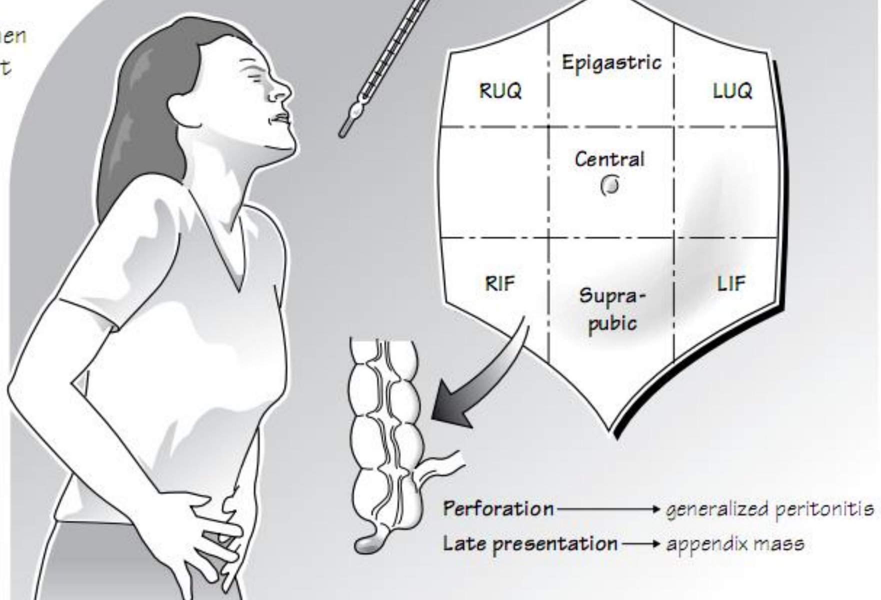
Important clinical questions to address
- Is the patient unwell?
- Is there hypovolaemia or shock?
- Is there an acute abdomen?
- Which organ(s) are producing the pain?

Appendicitis

History

- Periumbilical pain, then localizing to the right iliac fossa
- Nausea
- Vomiting





Examination

- Fever
- Tachycardia
- Pain, tenderness, rebound, guarding, McBurney's point
- Right-sided pelvic tenderness on PR examination

Differential diagnoses include:
- ectopic pregnancy
- pyelonephritis
- diverticulitis
- pelvic inflammatory disease and many others!

Appendicitis more likely
- Right iliac fossa: pain, tenderness, guarding

Appendicitis less likely
- Generalized pain
- Pain localized elsewhere in the abdomen
- Profuse diarrhoea
- Previous appendicectomy!

15

كثيرة هي حالات آلام البطن الحادة .. والطبيب المتميز يعرف كيف يشخص هذه الحالات جيدا

وسنعرض الكثير من الحالات التي تمر بنا وبها ألم بالبطن ... ولكن نبدأ بهذه الحالة اليسيرة وبنت في ال23 من عمرها غير متزوجة تشتكي فقط من ألم فجأة في الجنب الأيمن . فتقوم بسؤالها الكثير من الأسئلة التي تستبعد .. هل يوجد سخونية فتجيبك بالنفي

هل يوجد حرقان في البول .. فتجيبك بالنفي .. هل الألم متكرر.. فتقول كل شهر في نفس المعاد تقريبا ...ولكن الشهر الماضي كان في الجنب الأيسر ... فتسأل متي معاد الدورة الشهرية

فتجيبك لسه عليها اسبوعين يا دكتور.

تقريبا تكون التشخيص في رأسي لكن يجب أن أستبعد الأهم الان

يجب استبعاد الزائدة الدودية الملتهبة .. فتقوم بعمل اختباراتها المعروفة

Tenderness and rebound tenderness

Psoas sign

Obturator sign

No Nausea وهي شكوي مهمة جدا لمريض الزائدة الدودية

فتقوم بسؤالها سؤالا محرجا هل يوجد افرازات من الأسفل ..فتجيبك بكل كسوف نعم

إذا إنها الـــ متلشمرز

احم احم

هتلاقي حد بيقولك أنا فاكر حاجة زي كده بس دي يعني إيه إن شاء الله

بكل فخر تطمئن المريضة ألف مبروك ...((مش إنها حامل مش تستعجل .. اوعي تنسي إغنها آنسة)) تقولها ده يا آنسة آلام التبويض .. تحدث في منتصف الدورة الشهرية عندما تخرج البويضة من المبيض وتصنع بعض الالام ولن يستغر ال 12 ساعة

ويمكنك تناول أي مسكنات

عرفتوا يعني إيه المتلشمرز

Mittelschmerz : sudden onset of right or left lower quadrant pain with ovulation , copious mucoid vaginal discharge

ودمتم سالمين

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أم تحمل طفلتها في الثالثة من عمرها وتدخل محرجة وتخبرك .. لو سمحت يا دكتور البنت بتهرش من عند فتحة الشرج .. فبادرتها بالسؤال فيه حد من إخواتها أو من البيت بيهرش غيرها..فتجيبك بنعم

يجب علينا فحص الطفلة من أسفل حتي نطمئن ... فقمت بفحصها لأجد احمرارا وآثارا للهرش

فسألتها متي يزيد الهرش يا مدام .. فتجيبني بيزيد بالليل يا دكتور.

إذا إنها الدودة الدبوسية هي السبب في ذلك

لكن يجب عمل تحليل براز في الصباح الباكر نأخذ عينة من الطفلة ...لكن هذه الدودة معدين وتنتقل عن طريق

Egg ingestion

تلتصق بالأيادي عند الهرش أو التنظيف أو عن طريق ملايات أو ملابس داخلية ويضع الطفل يده في فمه وتبدأ دورة جديدة من

Auto infection

فتقوم بإعطائها علاجا وتخبرها بأن جميع أفراد الأسرة يجب أن يأخذوا من نفس العلاج

ويجب غلي جميع الملابس الداخلية والملايات

R/ Albendazole as a single oral dose to be repeated after one week

إن بي : وأنا في المطرية التعليمي وجدت حالة طفل في الرابعة من عمره يتعرض ل

Child sex abuse

الطفل عنده

Perianal condyloma

وهو مرض ينتقل عن طريق الاتصال الجنسي ؟؟؟!!!!!!!!!!!!!!

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تجري الأم بابنها وتصرخ ..فأسرعنا إليها لنجد أن ابنها البالغ من العمر سنتين يعاني من تشنجات .. إنها طوارئ إذا .. ماذا يجب علينا فعله الان....

يجب علينا أن نضعه في وضع الاستشفاء

Recovery position

ويجب أن نطمئن علي مجري الهواء ويجب أن نطمئن علي النفس ويجب أن نطمئن ألا يجرح المريض نفسه .. الحمد لله المريض يتنفس ولا وجود لأي علامات اختناق

بسرعة تم وضعه علي أكسوجين ووضعه في وضع الشفاء وتجهيز الشفاط

Suction of any secretion to keep airway patent

تحاول الممرضة تركيب كانيولا لكن الوضع صعب مع التشنجات

فتسأل الأم من امتي بيتشجنج يا ماما ...وهل حصلتله قبل كده وهل كان عنده سخونية

فأجابت .. من عشر دقائق يا دكتور والولد سخن خالص ودي أول مرة تيجيله

بالطبع أن تشك أنها تكون تنجات مصاحبة لارتفاع درجة الحرار

Febrile convulsion

لا وقت لتركيب كانيولا الان....طلبت من الممرضة نيوريل .. وسنذكر الاسم التجاري هنا لأنه متوفر في المستشفيات وهو عبارة عن

Diazepam

سحبت منه 1سم وأكملت باقي السرنجة ب محلول ملح وكشفت عن بنطاله

وأعطيته الحقنة شرجيا ((بالطبع بعد إزالة الإبرة))

RECTALLY

يمنع إعطائه عن طريق الحقن بالعضل ... فقط عن طريق الوريد وعن طريق الشرج

في غضون دقيقة توقفت التشنجات

لكن لابد من عمل شئ آخر مهم جدا وهو خفض درجة الحرارة.. فقمت بعمل كمادات ماء فاترة من الصنبور ..... وقمت بإعطائه لبوسة

Paracetamol pediatric suppository

حتي تنخفض الحرارة

لا يقف دوري عند هنا يجب علي معرفة سبب ارتفاع الحرارة وإعطائه العلاج المطلوب هنا

تذكروا فقط هذا الكلام فسوف تواجهوه كثيرا

لنتكلم عن هذا المرض بعض الشئ

يأتي غالبا للأطفال من عمر 6 شهور لعمر 6 سنوات بسبب ارتفاع في درجات الحرارة وغالبا يأتي مرة واحدة مع كل ارتفاع للحرارة في المرض الواحد

وغالبا تكون التشنجات لمدة أقل من ربع ساعة وتكون

Self limited

ولا يكون مصحوبا بأي علامة من علامات الـ

Lateralization

وبالتالي غالبا لا نحتاج لعمل رسم مخ .

EEG ElectroEnchephaloGram



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استدعوني لأقوم بكشف منزلي علي امرأة عجوز في الستين من عمرها تشتكي من ألم حاد جدااا في قدمها اليمني .. فقمت بالذهاب لها لأجدها تعاني من ألم حاد عند بداية الأصبع الكبير في القدم اليمني .. فسألتها عن الحرقان في البول فأجابت إنه شديد

وكشفت علي قدمها لأجد فيها احمرار وتورم وألم عند الضغط عليها

فسألتها عن تناولها للكثير من الأملاح ..فأجابت بنعم ... فسألتها هل يوجد ألم في القدم الأخري فأجابت بالنفي ... فسألتها هل تعالج من الضغط أو السكر ..

فأجابت بإنها تعاني من ارتفاع الضغط وتأخذ له دواء فطلبت رؤيته

لأجده عبارة عن

Captopril + Hydrochlocothiazide

فسألتها هل هذه أول مرة تعاني من هذا الموضوع

فأجابت بإنه متكرر عليها وكشفت من قبل عند طبيب عظام.. فأخبرتها هل قال لكي إنه النقرص؟؟؟؟ فأجابت متعجبة بإنه نعم قال لي إنه النقرص

وأعطاني العلاج وأنا ماشية عليه لكن الألم عاد مرة أخري ولا تدري ما السبب؟؟؟؟؟؟

فأخبرتها إن السبب بسيط جدا وهو علاج الضغط يحتوي علي مادة تزيد من ترسيب أملاح اليورات في المفاصل وهو السبب في عودة الألم وأن هذه المادة ممنوعة في مرضي النقرص

هل تذكرون يا سادة الأعراض الجانبية لهذه المادة

فقمت بتبديل علاج الضغط ليكون

Captopril only مع متابعة الضغط باستمرار لمعرفة مدي الاستجابة للدواء الجديد

وأمرتها بعمل كمادات مياه بادرة باستمرار علي قدمها اليمني

((يمنع وضع ثلج عليها حتي لا يؤدي لعمل

Necrosis to the skin as it leads to VC of blood vessels))

وأعطيتها علاج النقرص

R/NSAIDs injection and topical

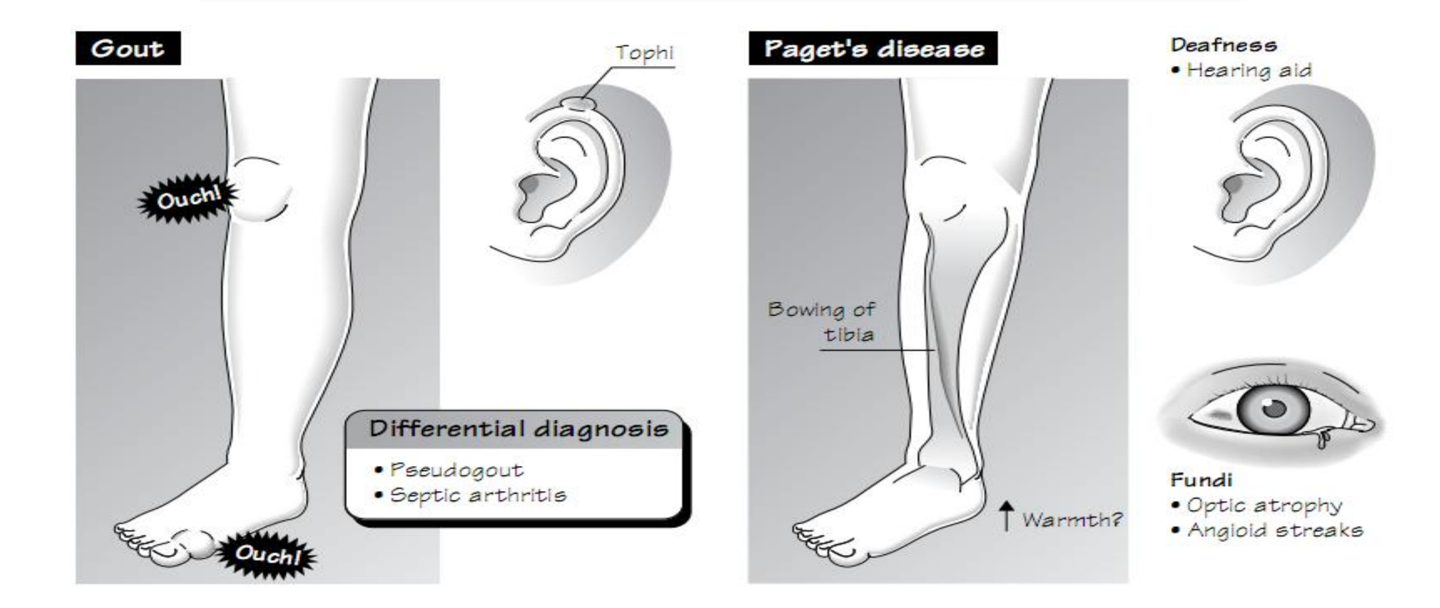
R/Uricosuric drugs like Cholchicine

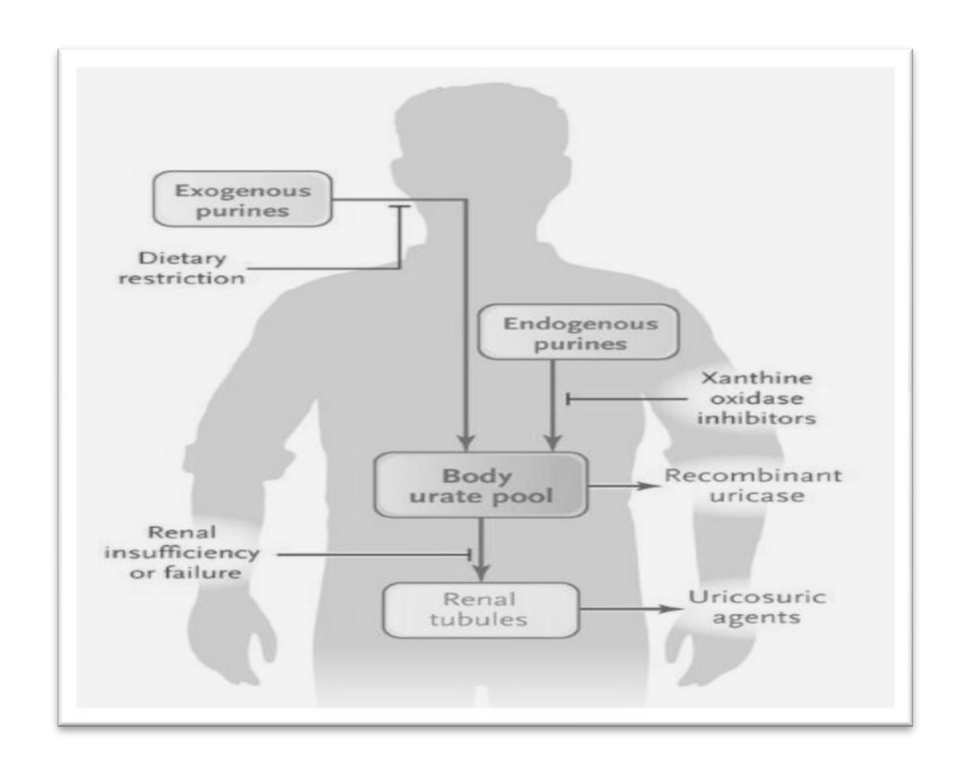
R/Allopurinol

ورأيتها بعد أسبوع فوجدتها بأحسن حال الحمد لله ووجدتها تدعوا لي كثييرا

فما أجمل العلم ... يعطيك الكثير والكثير لكن ما أجمل الدعوة الصادقة التي تأتي من أم عجوز

Side-effects Side-effects of thiazides and related diuretics include mild gastro-intestinal disturbances, postural hypotension, altered plasma-lipid concentrations, metabolic and electrolyte disturbances including hypokalaemia (see also notes above), hyponatraemia, hypomagnesaemia, hypercalcaemia, hyperglycaemia, hypochloraemic alkalosis, hyperuricaemia, and gout. Less common side-effects include blood disorders such as agranulocytosis, leucopenia, and thrombocytopenia, and impotence. Pancreatitis, intrahepatic cholestasis, cardiac arrhythmias, headache, dizziness, paraesthesia, visual disturbances, and hypersensitivity reactions (including pneumonitis, pulmonary oedema, photosensitivity, and severe skin reactions) have also been reported.





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امرأة في ال38 من عمرها من أقربائي تشتكي من ألم حاد بالمعدة يزداد مع الأكل ويقل مع استعمال الفوار المضادات للحموضة .لا تتعالج من أي مرض من ضغط أو سكر ولا تتناول مسكنات

قمت بالكشف الطبي عليها لأجد هناك

Epigastric Tenderness

No other tenderness in other parts in the abdomen

لم تتناول أي طعام من يوم بسبب الألم الشديد

سألتها هل فيه ترجيع لدم أو شئ بني اللون .. فأجابت بالنفي

وصلت للتشخيص تقريبا ..وهو التهابات بالمعدة

Gastritis

فقمت بتركيب محلول ملح وريدي ووضعت فيه

R/Zantak amp

R/ (Pantoprazole) vial,as Proton pump Inhibitorابحث عن الاسم التجاري

فشعرت بتحسن رائع ولله الحمد

لكن بعد ساعتين عاد الألم مرة أخري

فطلبت منهم تحليلا للبراز ولكن ليس كأي تحليل وإنما

Helicopacter Pylori Antigen in the stool

قاموا بعمل التحليل لأجد أن النتيجة إيجابية

إذا إنها البكتريا الحلزونية تعلن الحرب علينا

يجب علينا مواجهتها والاستعداد لها

هل تذكرون العلاج الثلاثي للقضاء عليها

Triple Therapy

(Proton pumb Inhibitors + Amoxicillin + Metronidazole)

لمدة أسبوعين مع الابتعاد عن المسبكات وعن أي أكل حراق أو أي

NSAIDs

وكلموني بعدها ولله الحمد تحسنت الحالة تماما

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أحكي لكم اليوم قصة لأوصل لكم فكرة .. طفل في السادسة من عمره أتي مع والده يشتكي من التهابات شديدة في الحلق وارتفاع في درجة الحرارة.. ووالده يشتكي إن الطفل لا يأكل ولا يلعب كما هو المعتاد منه.

قمت بالكشف الطبي الشامل عليه كما أفعل مع كل الحالات بعد أخذ تاريخ مرضي من الطفل ووالده .. ثم أقوم بالفحص علي المريض ككل ثم مكان شكواه..لأجد أنه يعاني من التهابات حادة في اللوزتين مع تضخم اللوزة الشمال عنده.

إذا إنها الـ

Acute tonsillitis

قمت بوزن الطفل وحسبت له الجرعة وكل شئ .. ووصفت لهم العلاج ومدة استعماله وهي الأسبوع حتي لو نزلت درجة الحرارة وحتي لو أتموا علبة المضاد الحيوي فيجب أن تكرر له الجرعة حتي تصل لمدة أسبوع

أتوا لي مرة أخري بعد 4 أيام ... ليقولوا لي أن الحالة لم تتحسن ..... من عادتي أني لا أجري مع كلام المريض والأهل يجب أن أستفسر جيدا عن العلاج .. ومن عادتي أيضا أني أؤمن نفسي دوما .. فكنت قد كتبت في الروشتة أن مدة العلاج بالمضاد الحيوي أسبوع وأضع خط تحت أسبوع وأؤكد علي المريض .. لأني أعلم أن أسباب مقاومة البكتريا هو أن يأخذ المريض جرعة أو اثنتين او يوم أو اثنين وتقل الحرارة فيتوقف عن العلاج وتقوم البكتريا باستعادة عافيتها وتقاوم العلاج ..وأيضا من أسباب انتشار الحمي الروماتزمية هو ما يحدث من سلوك الناس الخاطئ ويساعدهم في ذلك بعض الصيادلة .. مثلا يأتي المريض يطلب كبسولتين مضاد حيوي...ويتناولهم وبالتالي تقل السخونية وتقاوم البكتريا وتنتشر الحمي الروماتزمية

المهم نعود لحالتنا .. قست للطفل الحرارة لأجدها 38 ورأيت حلقه مرة أخري لأجد أن الالتهابات قلت كثيرا عن الأول وقل حجم اللوزة الشمال ..لكن الطفل يشتكي من بعض الاحتقان

فقمت بسؤال الطفل هل قل الاحتقان عن الأول يا حبيبي .. فيقول آه يا دكتور قل عن الأول ... بينما أمه كانت تقول إنه لا يوجد تحسن...فقلت لها أنا متأكد إنك مش أعطيتيه الدواء اليوم ..فقالت نعم أنا مش استعملته من يومين .. لأني بأعتمد علي الحقن وبتجيب نتيجة علي طول

الحمد لله ظهر الحق

كن واثقا في الله عز وجل أولا ثم في علاجك طالما أنت شخصت بتوفيق الله المرض وحسبت له الجرعة وأوضحت للمريض كل شئ فكن واثقا ودايما ((حقق مع المريض وأكد الكلام عليه))

سقت لكم هذه الحالة لأقول لكم كونوا واثقين من أنفسكم في الحق

ولا عيب أن تقولوا ليست حالتي .. ولا عيب أن تتراجع عن قرار أو علاج لتوضح الأحسن

وأخبركم بشئ .. لا تجعل سعر الدواء يوقفك عن كتاباته طالما هو المطلوب للعلاج والأقل منه لا يأتي بنفس النتيجة .. أهم شئ هو أن يكون المريض وأهله مقتنعين بك وبكلامك الصادق ((وليس الكاذب)) كما يفعل الكثيرون من الأطباء للأسف ... يُفتون ويَفِتّون ويخترعون ويؤلفون وينسون أن الله مطلع عليهم

ودوما اقرأوا وتعلموا وابحثوا علموا أنفسكم وتعلمون ممن هم أكبر منكم ممن تثقون فيهم واعلموا أنه لا كبير علي العلم .. ولابد أن تقرأوا يوميا في الطب

Learn How To Learn

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أتي الأب حاملا ابنته التي تبلغ من العمر 3 سنوات ومعهم الأم .. ليخبروني أن الطفلة كانت تعاني من أسبوع من سعال وارتفاع طفيف في درجة الحرارة .. ولأن الأهل في كثير من الأحيان يعتقدون أنهم جيدون ويخافون علي أبنائهم .. ولأنهم كشفوا من قبل عند الأستاذ فلان الفلاني وأعطاهم العلاج والحمد لله يسر الله الشفاء علي يديه ... فالأب تفضل غير مشكور علي فعلته .. بأن كرر للطفلة علاج الأستاذ القديم ...

وكان العلاج عبارة عن كورتيزون ((بريدسول)) ومضاد حيوي ((أزيثروميثين)) وخافض للحرارة ... فسألتهم عن سبب البريدسول فأخبروني إنها كان عندها حساسية من زمااان وكانت ماشية عليه ... المهم سألتهم أخبارها إيه مع الأكل واللعب .. فاجابوا إنها لا تأكل ولا تلعب زي زمان...

ولا أخفيكم سرا أنا كنت خائفا جدا من منظر الطفلة فهي كما يقولون بالانجليزية

The Baby isnot Doing Well

Has Mask face , she is deadly quite but befor when doctor come near here she cry and become aroused

فقمت بالكشف عليها لأجدها كما قلت لكم سابقا .. مع ارتفاع في الحرارة مع

Mild tachypnea , congested throat

إنها اللوزتين ... لكن ما سبب هذه الحالة ....لماذا هذه الطفلة غير عادتها؟؟؟؟

إنه الكروتيزون .. ظلت تتناوله 3مرات يوميا بدون غطاء من مضاد حيوي قوي

فأضعف مناعتها وقلل من مقاومة الجسم للبكتريا فأدي لهذه الحالة

فأعطيتها العلاج بعد حساب جرعتها علي حسب وزنها.

وتأتي الحالة ولله الحمد بعدها بأربعة أيام وقد تحسنت تماما

وكان العلاج عبارة عن الجيل الرابع من

Ceohalosporin + amoxicillin and claivulinic acid + analgesic antipyretic anti inflammatory

لماذا أخبركم بهذه الحالة؟؟؟؟ حتي تأخذوا تاريخ مرضي جيدا من الحالة

لا تكتفوا فقط بكلام المريض أكدوا عليه مرة واثنين وثلاثة ... واسألوا عن كل شئ

Good Physician Who takes a Good History and do a Good Examination and request the only needed investigation.

وأخبرتكم بهذه الحالة أيضا حتي تنبهوا علي المرضي عدم استعمال أي دواء بدون استشارة الطبيب .. وعند كتابة الكورتيزون يجب حساب الجرعة وكتاب مدة الاستخدام للمريض علي الروشتة والتنبيه عليه بعدم تكرار العلاج من تلقاء نفسه ويجب عليه الانتهاء منه في المدة المحددة . فالطبيب أخطأ بعدم توضيح مدة الاستعمال وعدم تكرار الدواء.

وستجدون هذا الكلام أيضا بخصوص مضادات الاحتقان الموضعية

Local decongestant

يجب استعمالها من مرتين لـ ثلاث مرات في اليوم ولمدة لا تزيد عن 3-4 أيام ويمنع تكرارها

حتي لا تقلب بأعراض جانبية شديدة عند المريض وهي

Chemical rhinitis

عودوا أنفسكم علي حساب الجرعة بالالة الحاسبة وعلي حسب وزن الطفل وكتابة كل شئ علي الروشتة وبعد كتابته يجب عليكم إخبار المريض بما يجب فعله

مثلا عندما تستعملوا الملطفات الجلدية مثل الكالاميل ... هذه مستحضرات مثل أدوية الشراب في نفس الزجاجة .. بعض المرضي شرب هذه المستحضرات بدلا من استعمالها علي الجلد .. لن الطبيب لم يوضح للحالة طريقة الاستعمال

مثل أيضا طريقة استعمال بخاخات جساسية الصدر هناك بعض المرضي معرفتهم الطبية قليلة خصوصا لو في قري بعيدة...يستعمل المريض البخاخة مثل زجاجات البارفانات ؟؟ ولا يستعملها بطريقة صحيحة

ويجب عليكم توضيح طريقة استعمال البخاخة حتي لا يفقد المريض أثررها عن طريق أن يضغط المريض علي البخاخة وهو يأخذ نفس عميق ووبعدها يكتم نفسه قدر استطاعته حتي يأخذ أقصي استفادة منها

كل هذا يأتي بالقراءة والمعرفة والتجربة والخبرة وسؤال من هو أكبر منك

ولا تجعل مرضاك حقلا لتجاربك عن جهل.

وتذكروا أن هذا واجبكم .. وحق المرضي عليكم .. دوما اجعلوا المرضي مثل أهليكم وانظروا هل تحبون أن يعامل أحد أهليكم مثل هذه المعاملة؟؟؟؟؟

22

كنت منهكا جدا هذا اليوم .. لكن هذه الحالة نشطت من ذهني الذي يحتاج الان لكثير من النوم

دخلوا علي بطفل حديث الولادة عمره 10 أيام . ليخبروني إنه عنده أكثر من مشكلة .. إن الطفل بيرجع علي طول .. وإنه عنده صفراء من أول يوم ولادة !!!!!!!!! وإنهم كشفوا عند طبيب آخر من قبل لكنه لم يستجب للعلاج وأراد أن يعطيني الروشتة الخاصة بالطبيب الاخر لكني رفضت الان حتي لا يتم توجيهي بتشخيص قديم ... ففضلت أن أبدأ مع الحالة من البداية

سألته الصفراء بدأت متي ؟ فقالي من أول يوم ولادة .

فبادرته بسؤال تعرف فصيلة والدته ؟ فقال لي لا أعرف .. وأمه لم تكن موجودة لأنه ولدت الطفل قيصريا .. فسألته مرة أخري هل دخل حضانة بعد لما اكتشفتوا الصفراء فقال لي لا.. فتعجبت جدا ((كل هذا ولم أري الطفل ولم أضع يدي عليه))

وسألتهم هل ده أول طفل ليهم فأخبروني إن له أخ يكبره ب3سنوات ولم يعاني من الصفراء

المهم استغربت جداااا وسألته عملتوا إيه فأخبرني إن الطبيب الأول قال لهم يضعوه تحت لمبة فلوروسينت 24 ساعة كل هذا من أول يوم ولادة حتي اليوم العاشر .. وأعطاهم علاجا للصفراء

تركت كل شئ وقمت بسرعة لأري الطفل لأري أين الصفراء ؟؟؟؟ فلو كان حقيقيا لكان هذا الطفل مريضا بشدة الان.. وسألتهم هل بيرضع كويس؟؟؟ فأجابوا نعم ؟؟ فتعجبت من هذا التناقض.

قمت لأجد أن الطفل لا يوجد به أي أثر للصفراء ..فسألته فين الصفراء اللي الدكتور قالكم عليها فأشاروا لأنفه ..فوددت عليها نقاط صفراء بحجم سن القلم .. وهذه النقاط يعرفها أي طبيب جلدية

ولأني ولله الحمد محب للجلدية وأقرأ فيها كثيرا عرفت الحالة وهي تنتج عن

Obstruction to sweat glands

وبتقعد مع الطفل فترة تصل لـ6 شهور وتختفي بعدها

فأخبرت الأهل أنه لا توجد صفراء أصلا

ولو كانت عنده من أول يوم كان هيكون محجوز في المستشفي وإن اللمبة النيون دي غلط جداا وغير مفيدة أصلا في حالات الصفراء.

لماذا سقت لكم هذه الحادثة

Please My Friend Donot Swallow The Diagnosis

Be Confident of your self IF YOU REALLY KNOW WHAT TO DO

ولا تكن مكابرا في الخطأ فلو أخطأت فقل أنك مخطئ واستشر من هو أكبر منك

وابحث وتعلم

وللأسف الطبيب الأول طبيب أمراض صدر وحساسية.

Mezoo

Notes

Dr Muhammad Barakat



السلام عليكم ورحمة الله وبركاته

في المجال الطبي دوما ينبغي أن تكون مرتب الذهن تعرف ماذا تفعل

لا تقوم بالكشف وفقط .. بل تكشف وتبحث عن أشياء وتستبعد أخري

ومن أهم الأساليب التي تساعد الطبيب علي هذه الأمور هي نظام

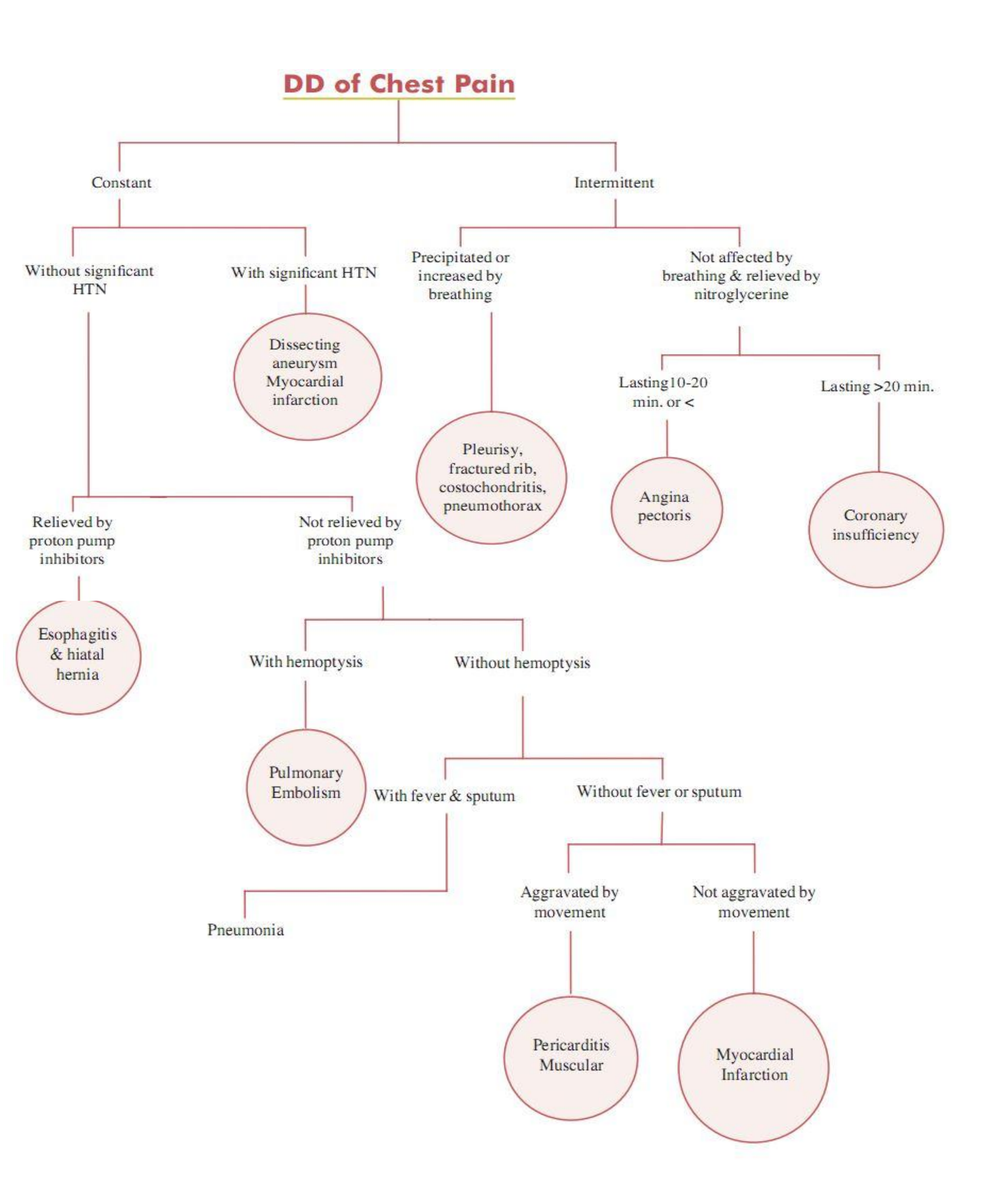
Algorithm

وهو ما سأقدمه لكم في هذه المذكرات

ستعرفون فيها كيف تتعاملون مع الحالات وكيف تفكرون فيها

لن أتكلم كثيرا لكن جرب أن تطبقها علي الحالات التي تأتي لك

وفقكم الله


DD of Chest Pain
Constant
Intermittent
Without significant HTN
With significant HTN
Dissecting aneurysm Myocardial infarction
Precipitated or increased by breathing
Not affected by breathing & relieved by nitroglycerine
Pleurisy, fractured rib, costochondritis, pneumothorax
Lasting 10-20 min. or <
Lasting >20 min.
Angina pectoris
Coronary insufficiency
Relieved by proton pump inhibitors
Not relieved by proton pump inhibitors
Esophagitis & hiatal hernia
With hemoptysis
Without hemoptysis
Pulmonary Embolism
With fever & sputum
Without fever or sputum
Pneumonia
Aggravated by movement
Not aggravated by movement
Pericarditis Muscular
Myocardial Infarction

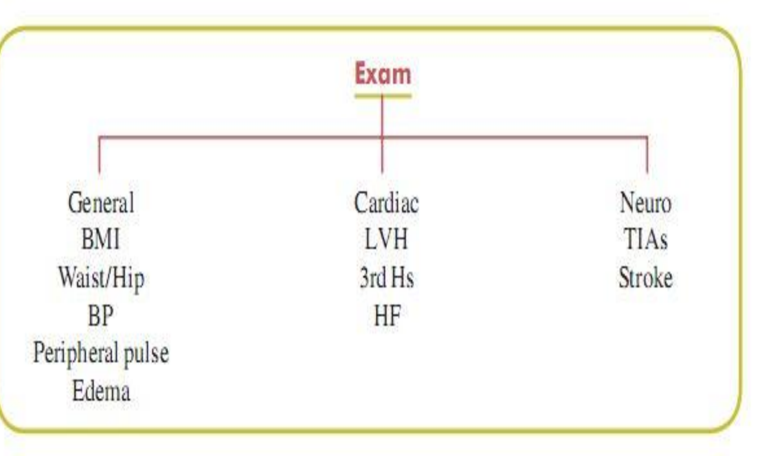
Secondary Prevention of MI

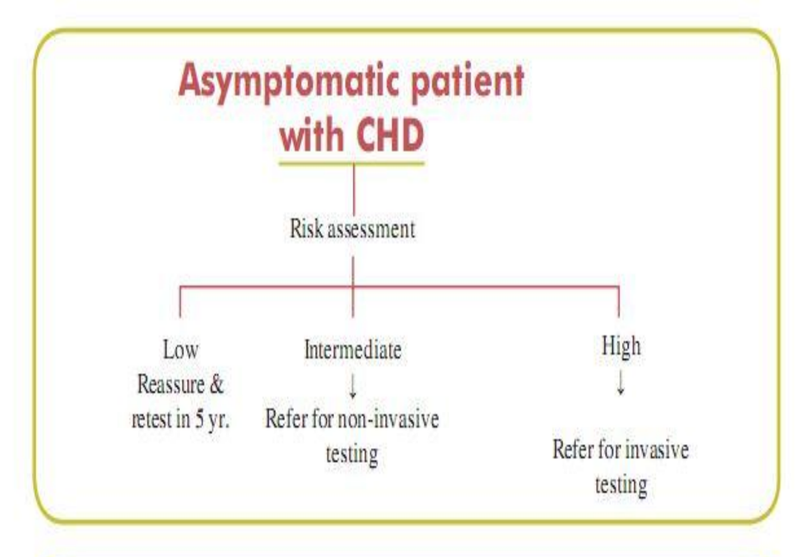
- Control risk factors
  - Stop Smoking
  - Dyslipidemia
    - Diet
    - Drugs
    - Target: LDL <100 mg/dL HDL > 35 mg/dL
  - Inactivity (Uncomplicated MI)
    - Gradual supervision
    - 2 wk: walk around
    - 4 wk: → 2 mile/d
    - 6wk: 2 miles in < 30 min/d
    - Return to sedentary work
    - Driving may be allowed
    - Resume sexual activity
    - 8 wk: return to light manual work
    - 3 mo: return to heavy manual work
    - Flying > 2 wk if can climb 1 floor without difficulty
- Drugs
  - B-blockers*
  - Aspirin (as mentioned before)
  - ACEI*

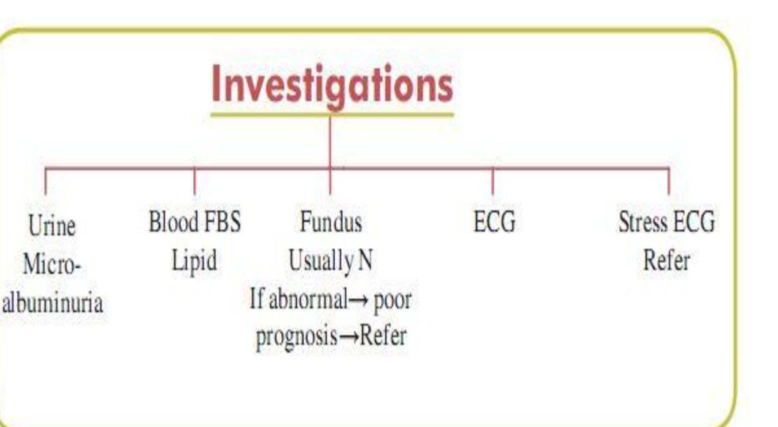
If Patient is presenting with Chest Pain of Angina

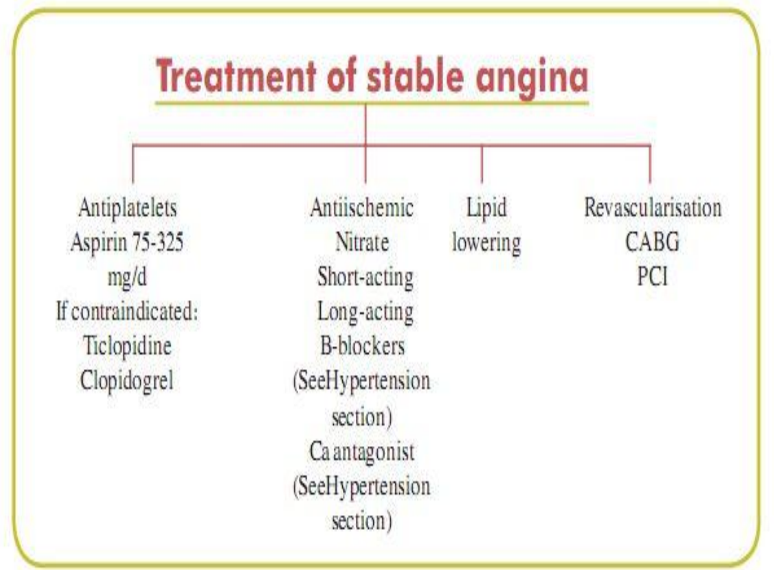
Sublingual nitrate tablet

- Relieved → Angina
- Not relieved → 2nd.tab after 5 min. → 3rd.tab after 10 min. → Not relieved → Chew & swallow aspirin 160-325 mg & Emergency transfer to hospital (unstable angina/MI)
- Not Relieved









Flow Chart Diagram For Treatment of stable angina

Algorithm for Causes

Systolic & Diastolic

Constant

Paroxysmal

Pheochromocytoma

Same or higher blood pressure in lower extremitie

Lower blood pressure in lower extremities

Coarctation of aorta

Flank mass

Hypernephroma, Hydronephrosis, Polycystic Kidney

No Flank mass

Abnormal urine sediment

Glomerulonephritis, Collagen Disease, Henoch-Schonlein Purpura, Chronic Nephritis

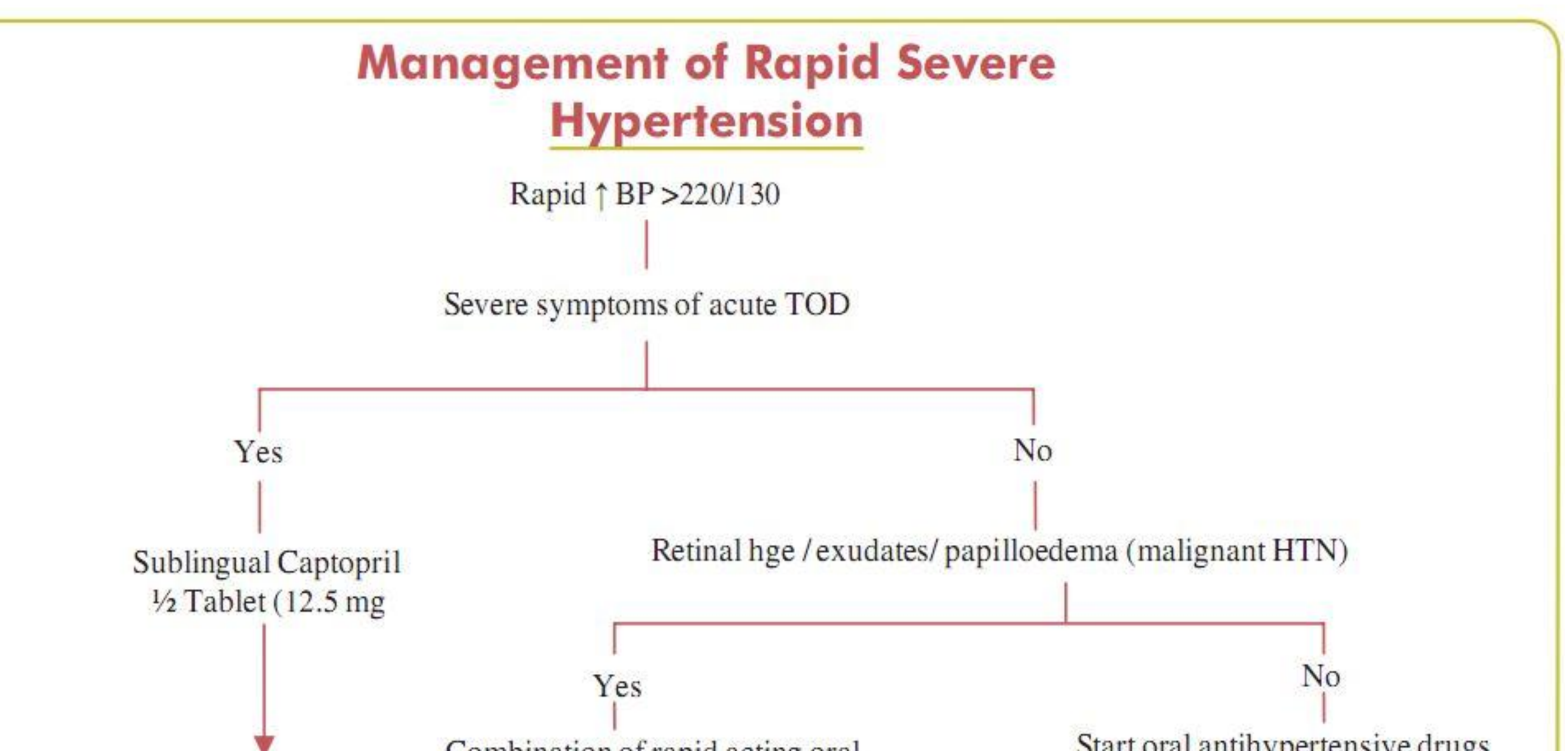
Normal urine sediment

Essential HTN, Cushing's syndrome, Aldosteronism, Some Pheochromocytomas

Systolic only

Hyperthyroidism, Aortic insufficiency, Atherosclerotic Aortitis

Flow Chart Diagram For Algorithm for Causes of Hypertension

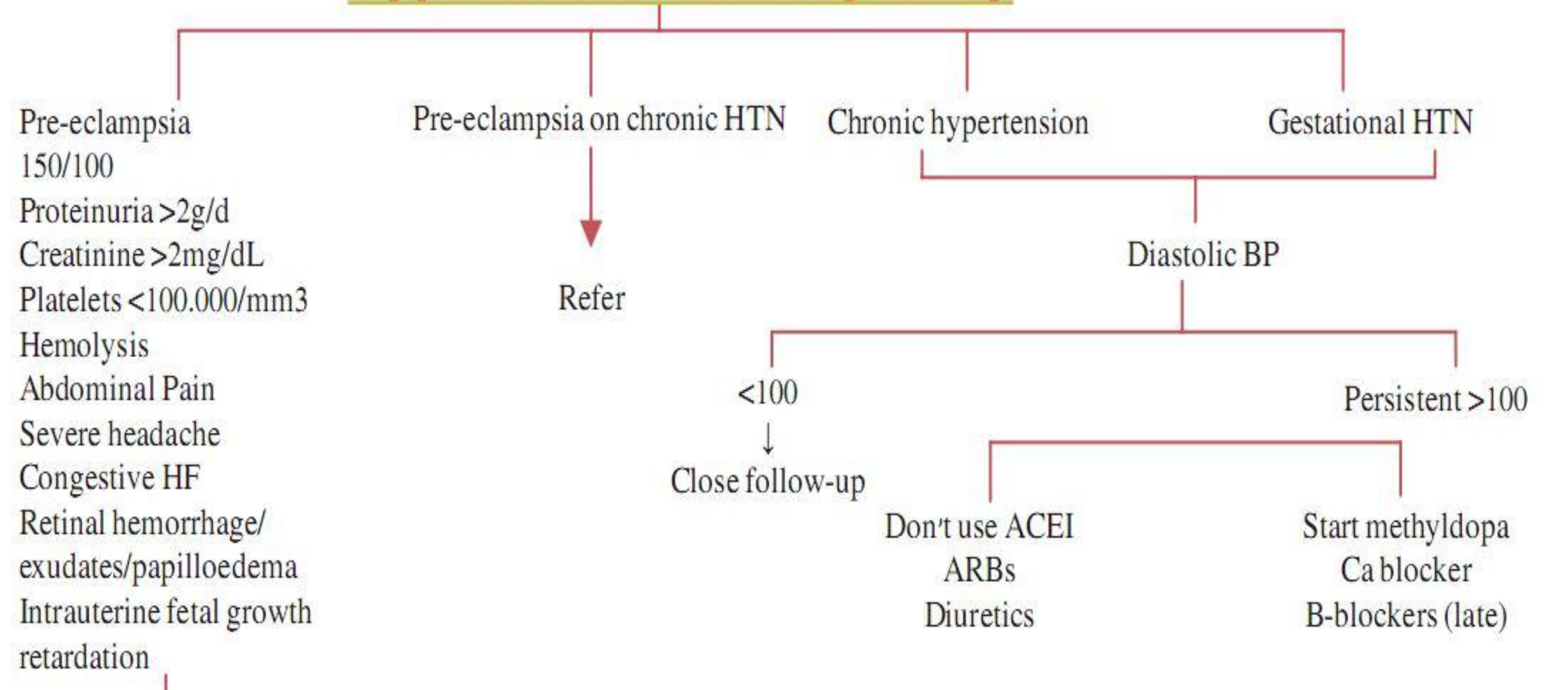


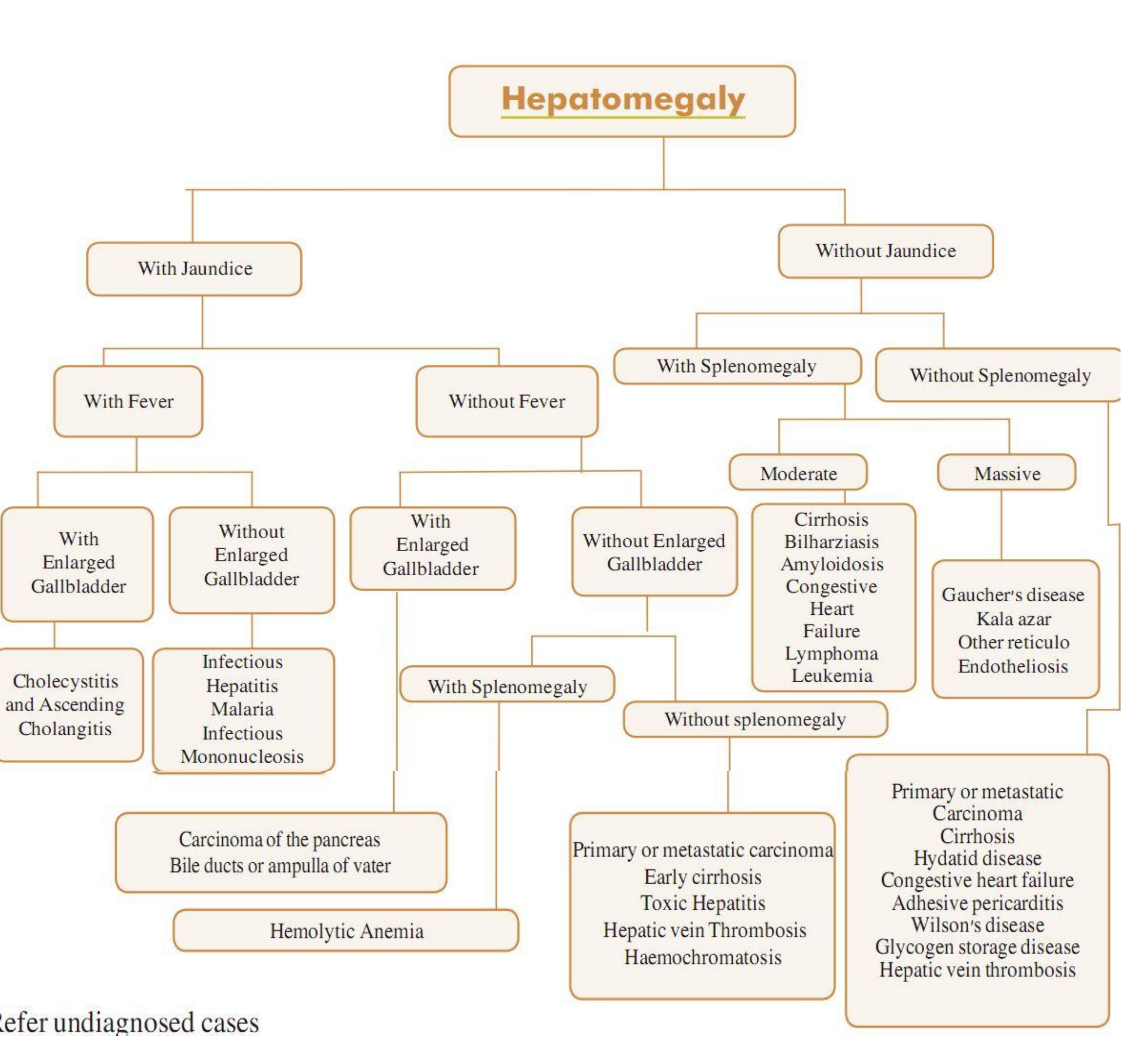
<table>
<tr><th>Drug</th><th>Dosage Form</th><th>Side-effects</th><th>Contraindication</th></tr>
<tr><td>Hydrochlorothiazide</td><td>tablets 25 mg</td><td rowspan="2">CVD: postural hypotension
Digestive: jaundice diarrhea, vomiting, constipation, gastric irritation, nausea, anorexia.
Hematologic: leukopenia, hemolytic anemia, thrombocytopenia.
Hypersensitivity
Metabolic: Electrolyte imbalance</td><td>- Sever renal & hepatic impairment
- Anuria.</td></tr>
<tr><td>Furosemide</td><td>Tablets 40 mg
Injection 40mg</td><td>- Liver cirrhosis
- Hypokalaemia
- Anuria.</td></tr>
<tr><td>Spirolactone</td><td>tablets 25mg</td><td>Digestive: Gastric bleeding, gastritis, diarrhea nausea and vomiting.
Endocrine: Gynecomastia
Hematologic: Agranulocytosis
Hypersensitivity</td><td>- Renal impairment
- Anuria.
- Hyperkalemia.</td></tr>
<tr><td>Captopril</td><td>tablets 25mg</td><td>- Cough, Rash
- Renal Failure
- Neutropenia
- Angioedema
- Taste impairment</td><td>- Bilateral renal artery stenosis
- Hyperkalaemia
- Neutropenia</td></tr>
<tr><td>Atenolol</td><td>tablets 50 mg</td><td rowspan="2">- Bradycardia
- Cold Extremities
- Tiredness
- Impotence</td><td rowspan="2">- Sinus bradycardia
- Heart block greater than first degree
- Cardiogenic shock</td></tr>
<tr><td>Propranolol</td><td>tablets 10 mg
& 40 mg</td></tr>
<tr><td>Nifedipine</td><td>tablets 20 mg</td><td>- Flushing
- Oedema
- Postural hypotension
- Headache</td><td>- Hypersensitivity</td></tr>
<tr><td>Deltiazem</td><td>tablets 60 mg capsules 90 mg, 120mg, 180mg</td><td>- Bradycardia/ heart block</td><td>- Heart failure, heart block
- Severe hypotension (less than 90 mm Hg systolic)</td></tr>
<tr><td>Methyldopa</td><td>tablets 250 mg</td><td>- Drowsiness during the first few weeks of therapy
- Fluid retention
- Headache
- Weakness</td><td>- Liver disease or cirrhosis
- Hypersensitivity</td></tr>
</table>

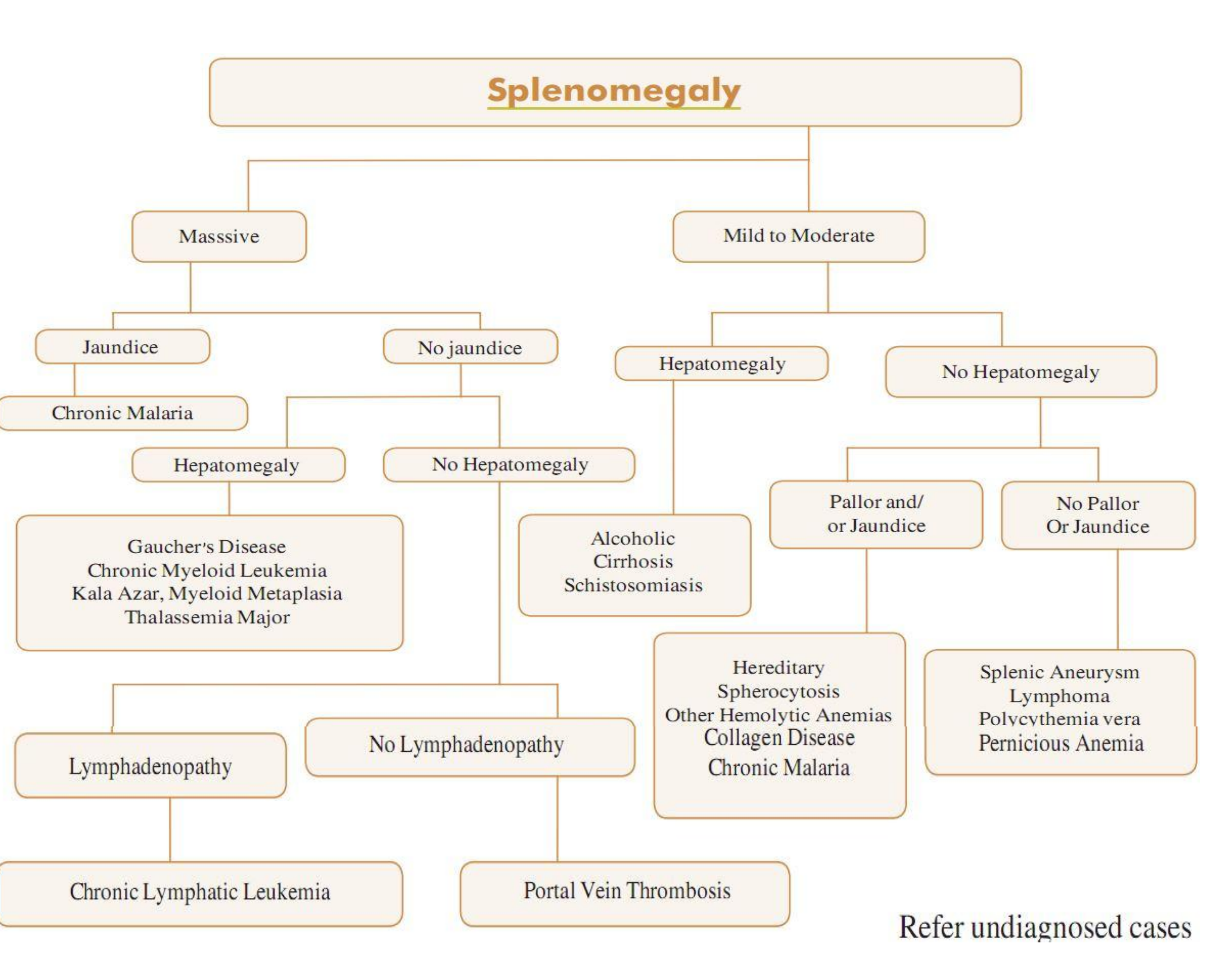
Table. 10: Treatment in Special Situations

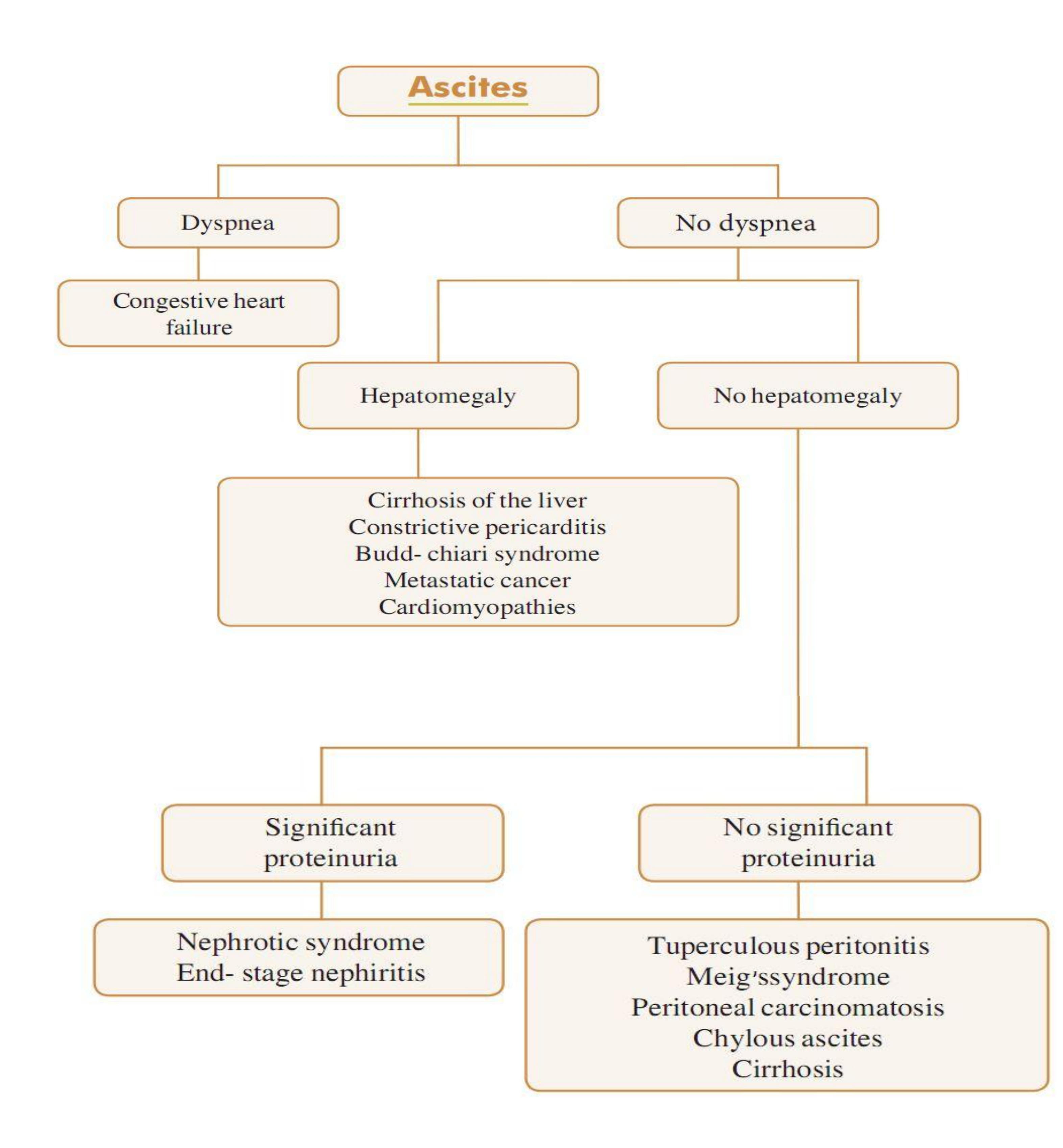
Conditions	Preferred drug	Problematic drugs
1.Diabetes Mellitus	ACE inhibiors,CA	B-blockers, high doses of diuretics
2.Systolic Heart Failure	ACE inhibiors,diuretics	B-blockers (except carvedilol),CA,diuretics
3.Diastolic Heart Failure	ACE inhibiors, B-blockers,CA	Diuretics
4.Angina	B-blockers, CA	Short acting dihydropyridine CA(e.g. nifedipine)
5.Myocardial Infarction	B-blockers, ACE inhibitors(with systolic dysfunction)	Short acting dihydropyridine CA(e.g. nifedipine)
6.Obstructive Lung Disease	ACE inhibitors	B-blockers, combined α & β -blockers
7.Renal Insufficiency	Diuretics, ACE inhibitors (if serum creatinine <3 mg/dl)	ACE inhibitors, A II receptor blockers, K-sparing diuretics
8.Pregnancy	Methyl dopa,B-blockers (in late pregnancy),hydralazine	ACE inhibitors, A II receptor blockers

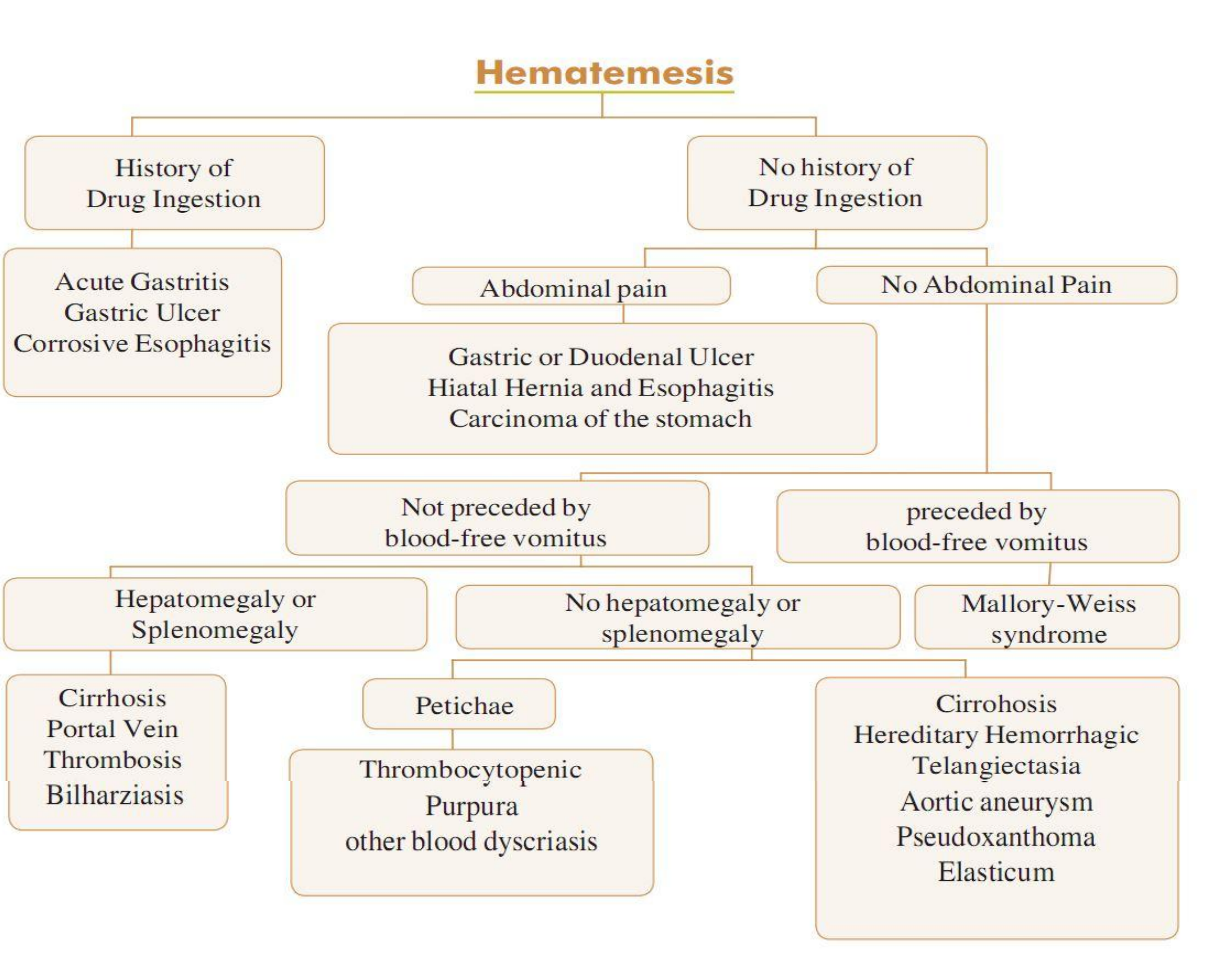
Hypertension & Pregnancy

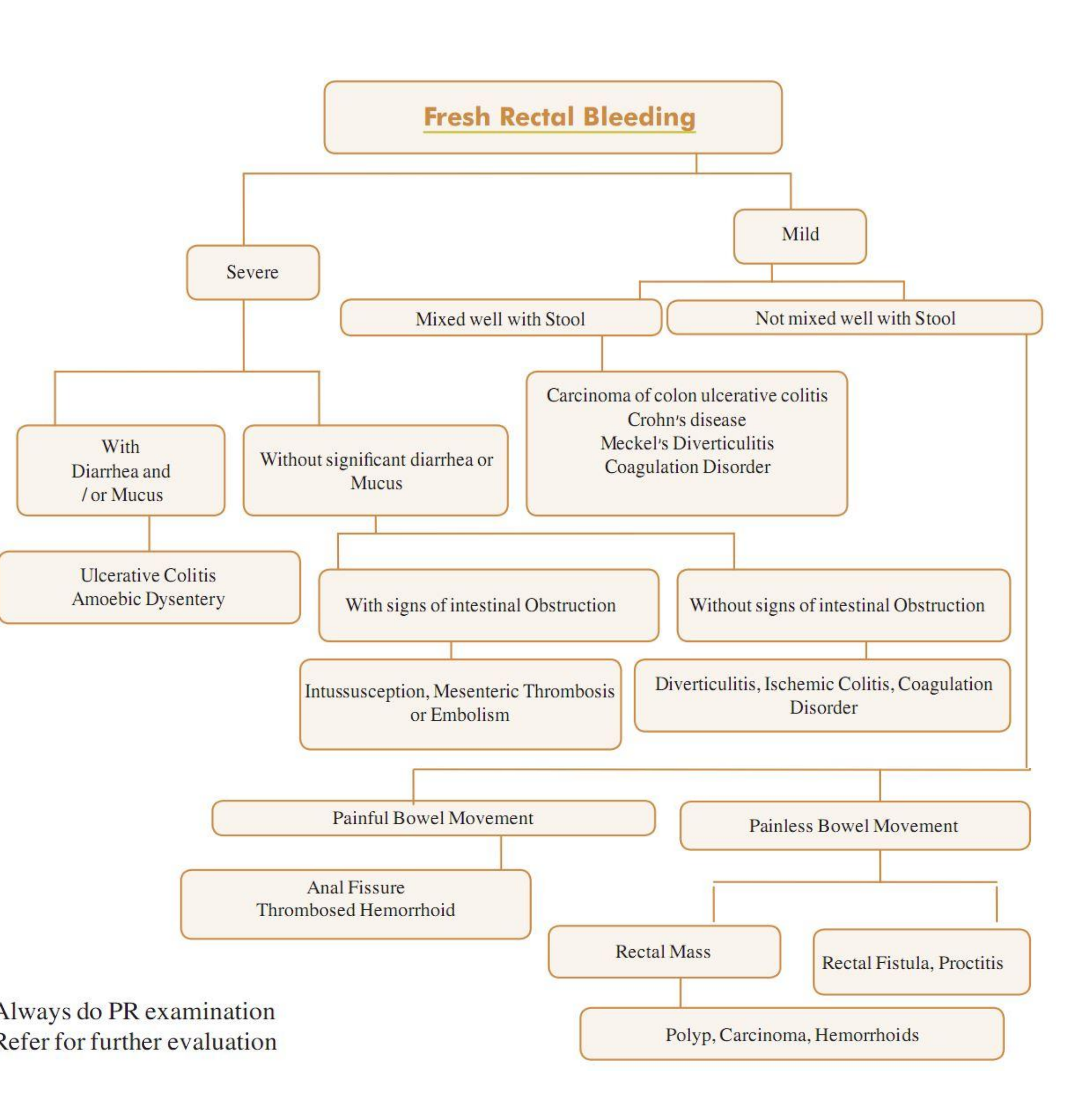


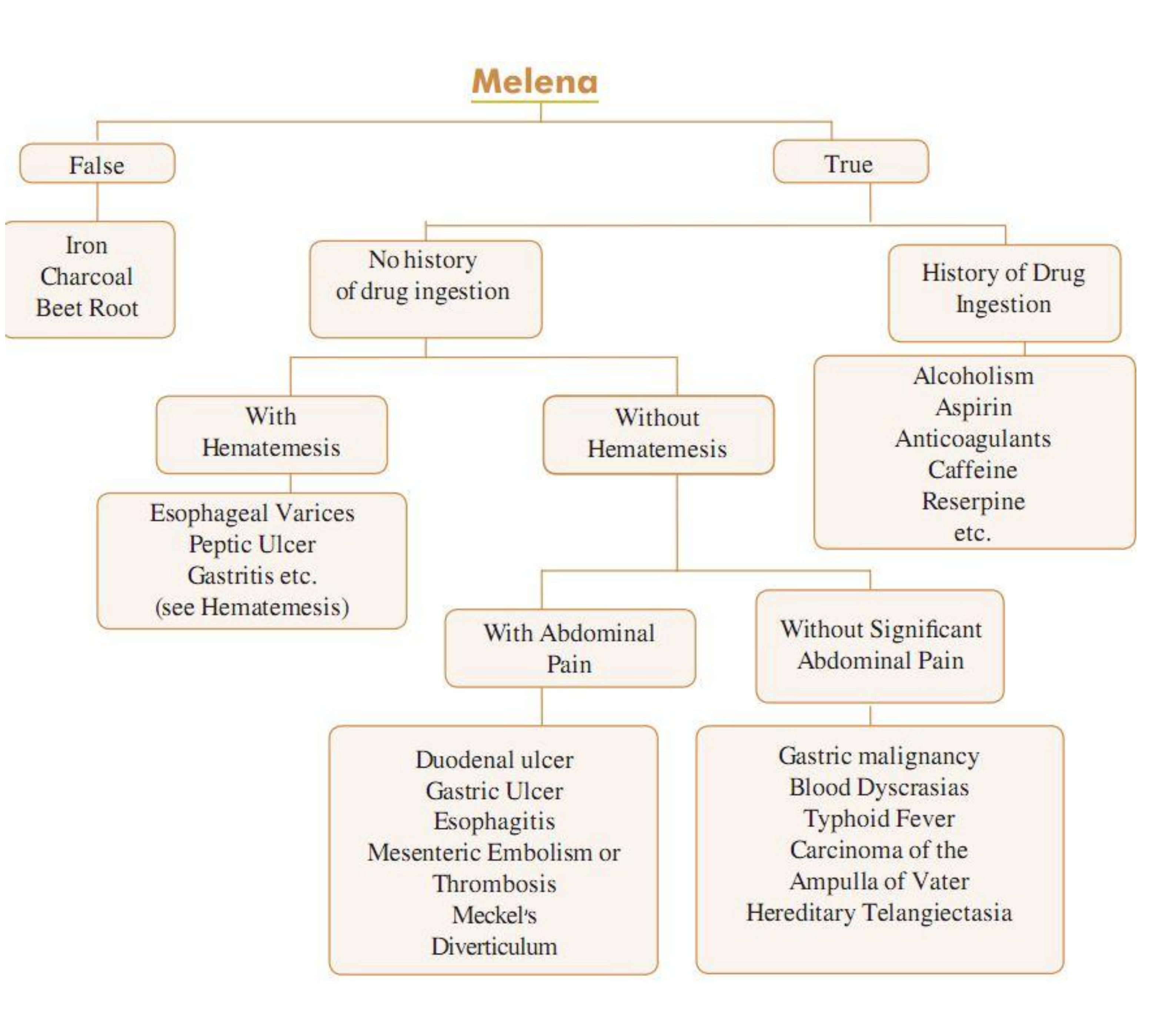

Hepatomegaly
With Jaundice
Without Jaundice
With Fever
Without Fever
With Splenomegaly
Without Splenomegaly
Moderate
Massive
With Enlarged Gallbladder
Without Enlarged Gallbladder
With Enlarged Gallbladder
Without Enlarged Gallbladder
Cirrhosis Bilharziasis Amyloidosis Congestive Heart Failure Lymphoma Leukemia
Gaucher's disease Kala azar Other reticulo Endotheliosis
Cholecystitis and Ascending Cholangitis
Infectious Hepatitis Malaria Infectious Mononucleosis
With Splenomegaly
Without splenomegaly
Carcinoma of the pancreas Bile ducts or ampulla of vater
Hemolytic Anemia
Primary or metastatic carcinoma Early cirrhosis Toxic Hepatitis Hepatic vein Thrombosis Haemochromatosis
Primary or metastatic Carcinoma Cirrhosis Hydatid disease Congestive heart failure Adhesive pericarditis Wilson's disease Glycogen storage disease Hepatic vein thrombosis
efer undiagnosed cases

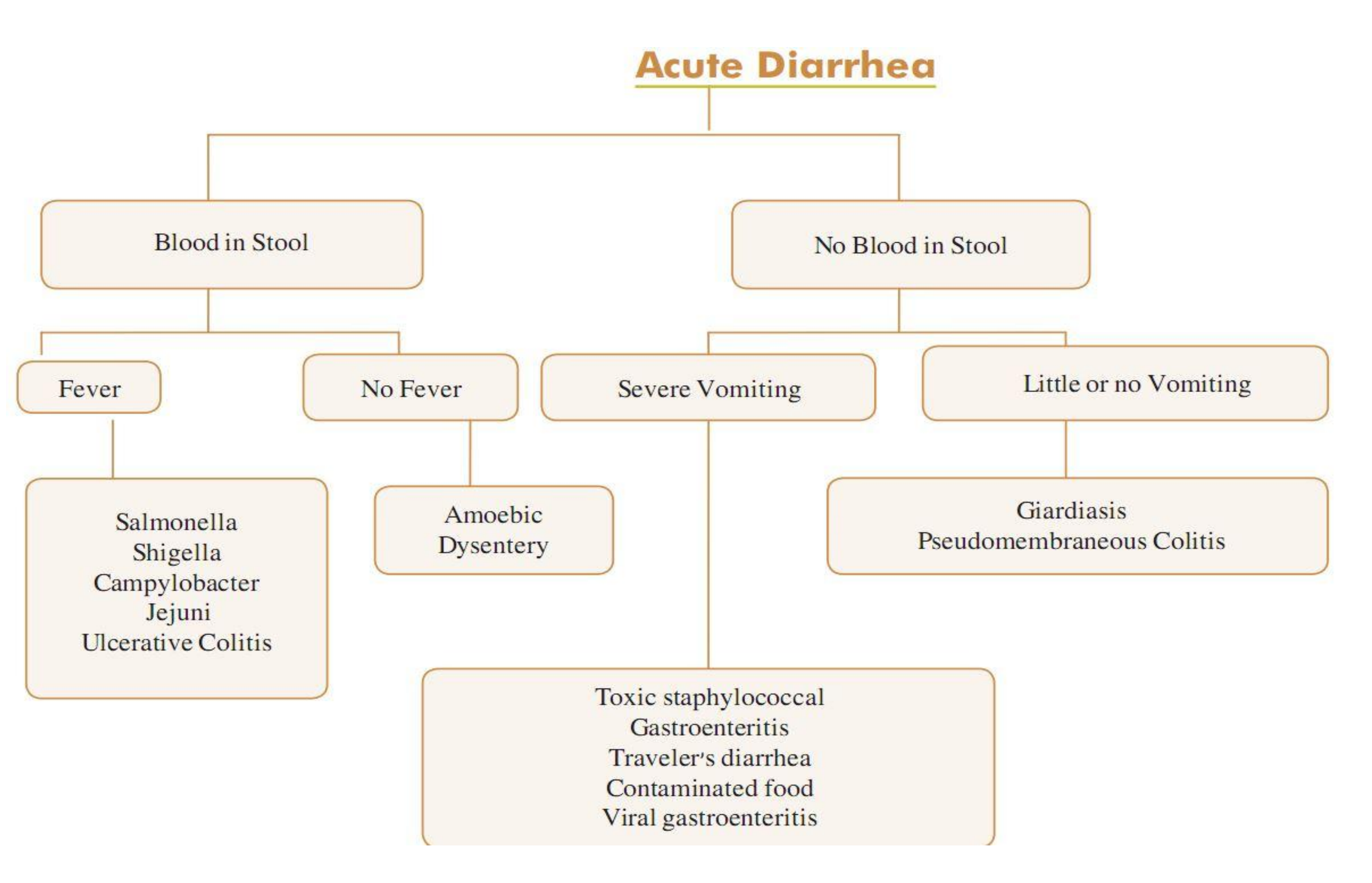

Splenomegaly
Masssive
Mild to Moderate
Jaundice
No jaundice
Chronic Malaria
Hepatomegaly
No Hepatomegaly
Gaucher's Disease
Chronic Myeloid Leukemia
Kala Azar, Myeloid Metaplasia
Thalassemia Major
Lymphadenopathy
No Lymphadenopathy
Chronic Lymphatic Leukemia
Portal Vein Thrombosis
Hepatomegaly
No Hepatomegaly
Alcoholic
Cirrhosis
Schistosomiasis
Pallor and/
or Jaundice
No Pallor
Or Jaundice
Hereditary
Spherocytosis
Other Hemolytic Anemias
Collagen Disease
Chronic Malaria
Splenic Aneurysm
Lymphoma
Polycythemia vera
Pernicious Anemia
Refer undiagnosed cases

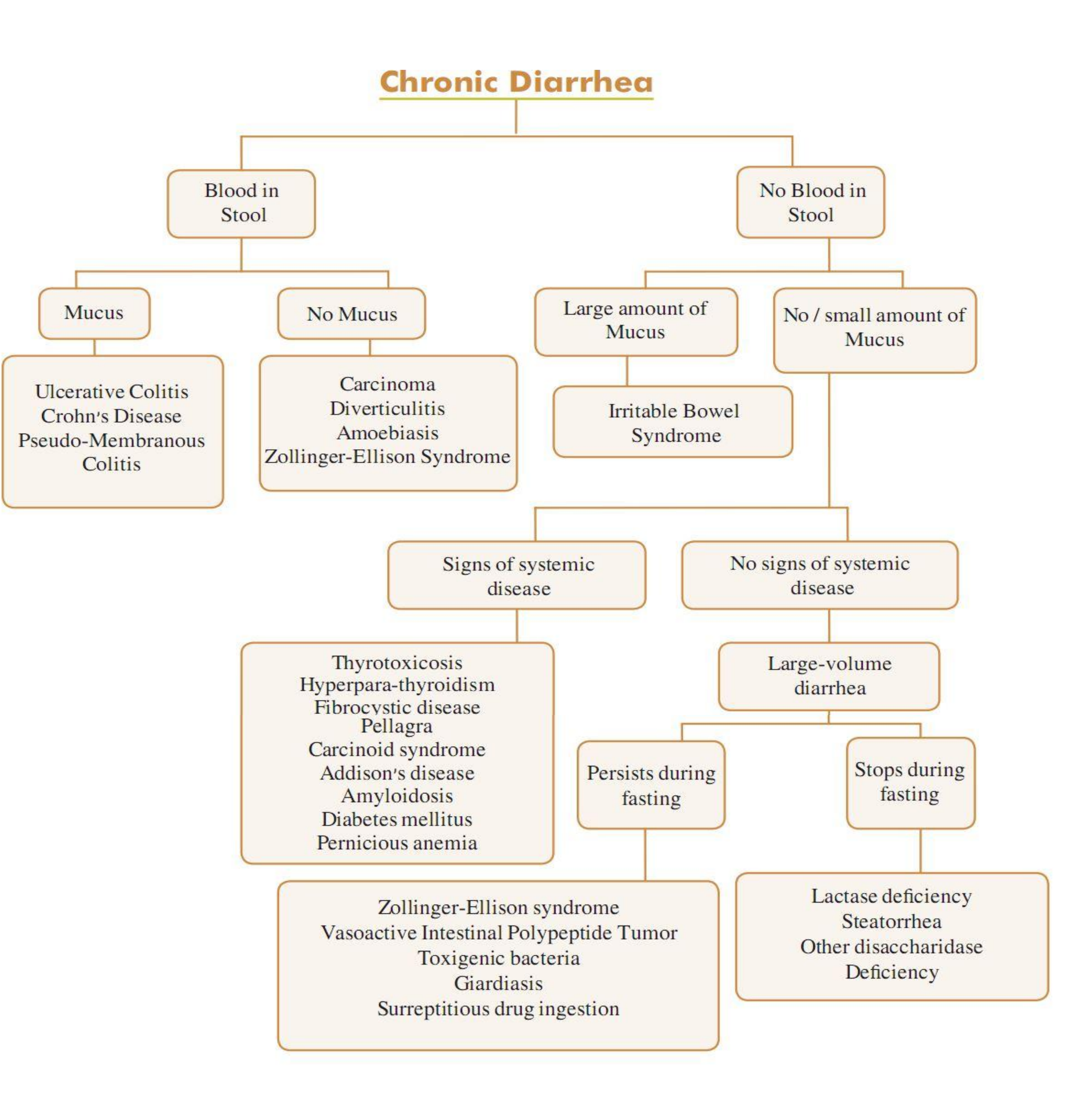

Ascites
Dyspnea
No dyspnea
Congestive heart failure
Hepatomegaly
No hepatomegaly
Cirrhosis of the liver
Constrictive pericarditis
Budd- chiari syndrome
Metastatic cancer
Cardiomyopathies
Significant proteinuria
No significant proteinuria
Nephrotic syndrome
End- stage nephiritis
Tuperculous peritonitis
Meig'ssyndrome
Peritoneal carcinomatosis
Chylous ascites
Cirrhosis

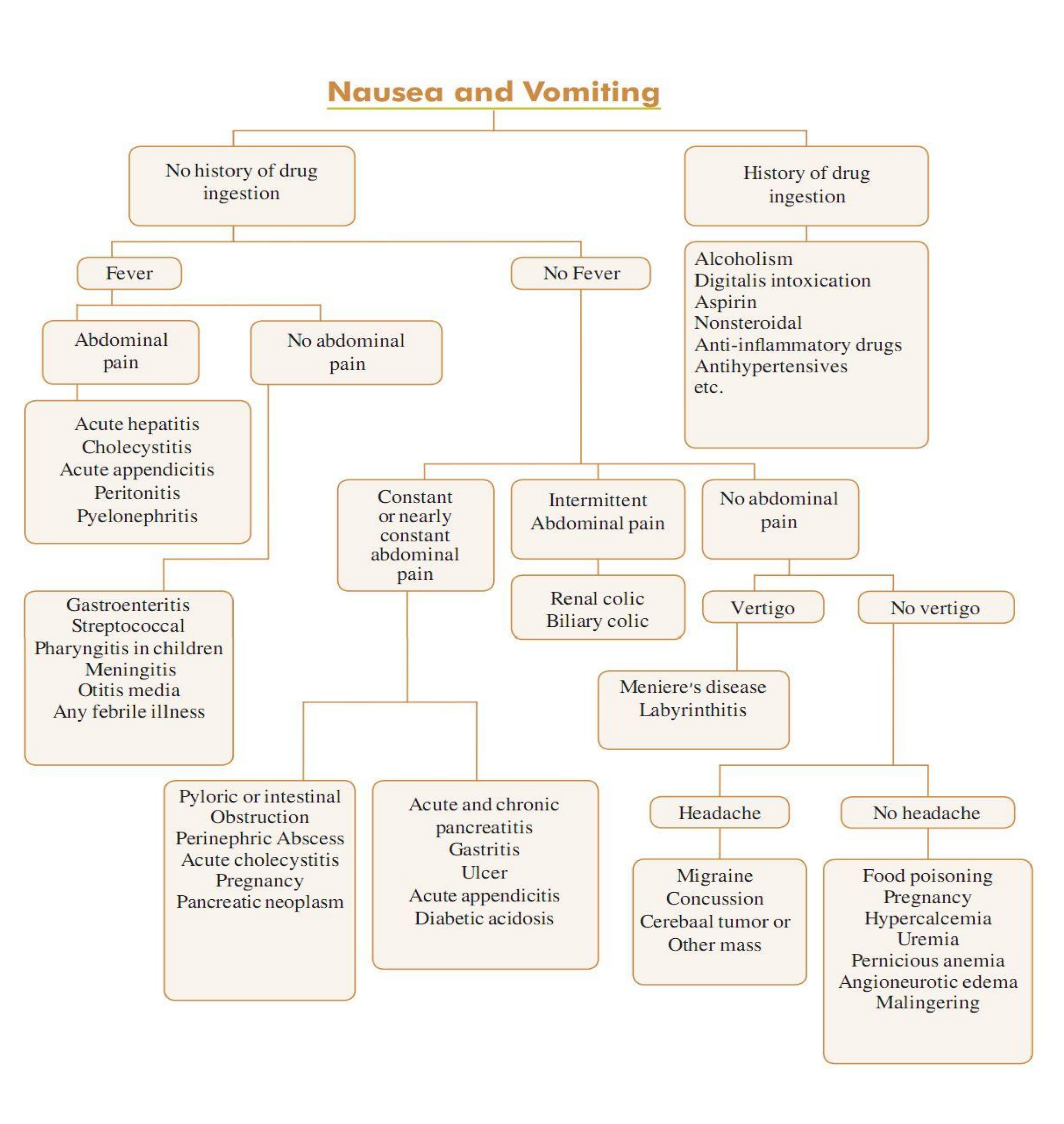

Hematemesis
History of Drug Ingestion
No history of Drug Ingestion
Acute Gastritis
Gastric Ulcer
Corrosive Esophagitis
Abdominal pain
No Abdominal Pain
Gastric or Duodenal Ulcer
Hiatal Hernia and Esophagitis
Carcinoma of the stomach
Not preceded by blood-free vomitus
preceded by blood-free vomitus
Hepatomegaly or Splenomegaly
No hepatomegaly or splenomegaly
Mallory-Weiss syndrome
Cirrhosis
Portal Vein Thrombosis
Bilharziasis
Petichae
Thrombocytopenic Purpura
other blood dyscriasis
Cirrohosis
Hereditary Hemorrhagic Telangiectasia
Aortic aneurysm
Pseudoxanthoma Elasticum

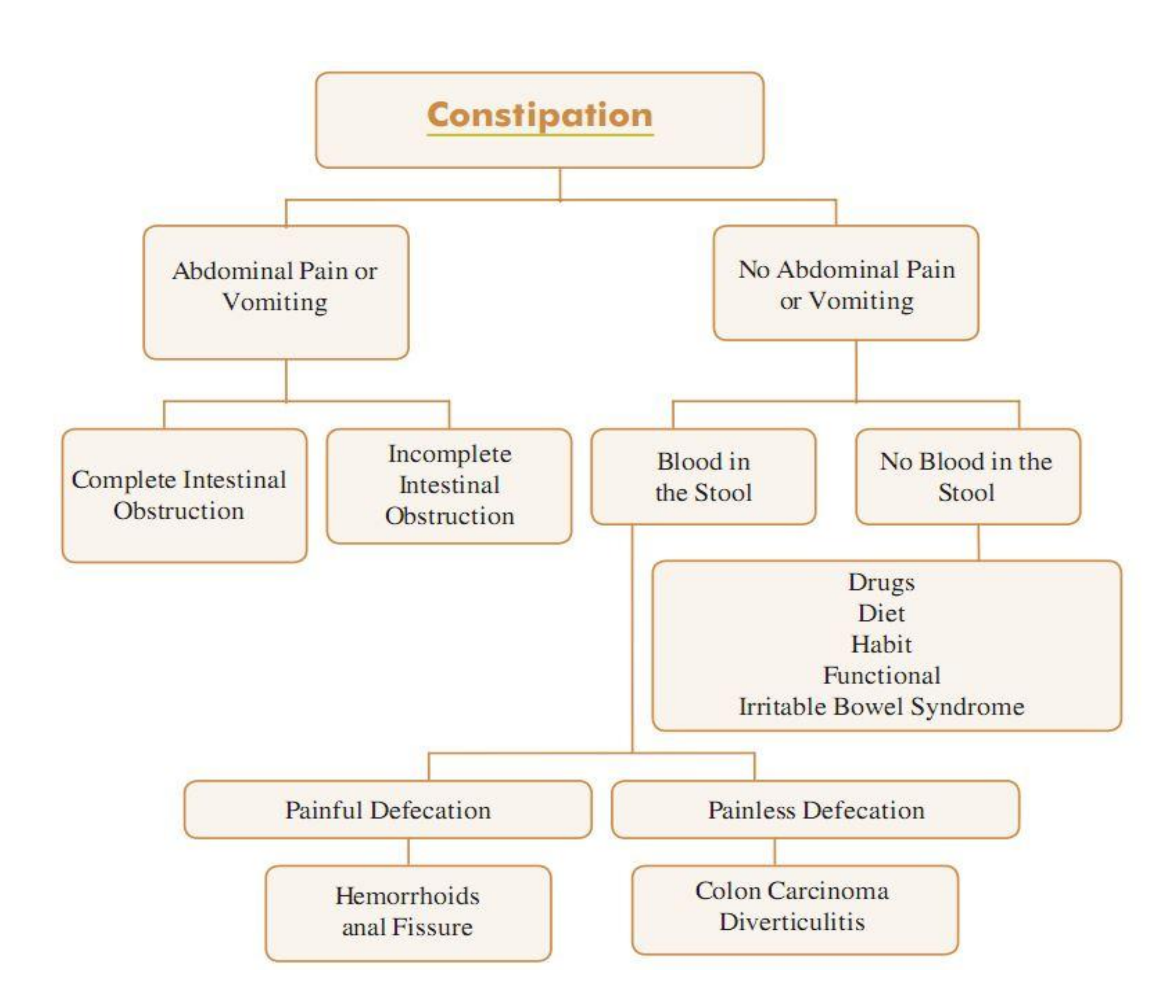

Fresh Rectal Bleeding
Severe
Mild
Mixed well with Stool
Not mixed well with Stool
Carcinoma of colon ulcerative colitis
Crohn's disease
Meckel's Diverticulitis
Coagulation Disorder
With
Diarrhea and
/ or Mucus
Without significant diarrhea or
Mucus
Ulcerative Colitis
Amoebic Dysentery
With signs of intestinal Obstruction
Without signs of intestinal Obstruction
Intussusception, Mesenteric Thrombosis
or Embolism
Diverticulitis, Ischemic Colitis, Coagulation
Disorder
Painful Bowel Movement
Painless Bowel Movement
Anal Fissure
Thrombosed Hemorrhoid
Rectal Mass
Rectal Fistula, Proctitis
Polyp, Carcinoma, Hemorrhoids
Always do PR examination
Refer for further evaluation

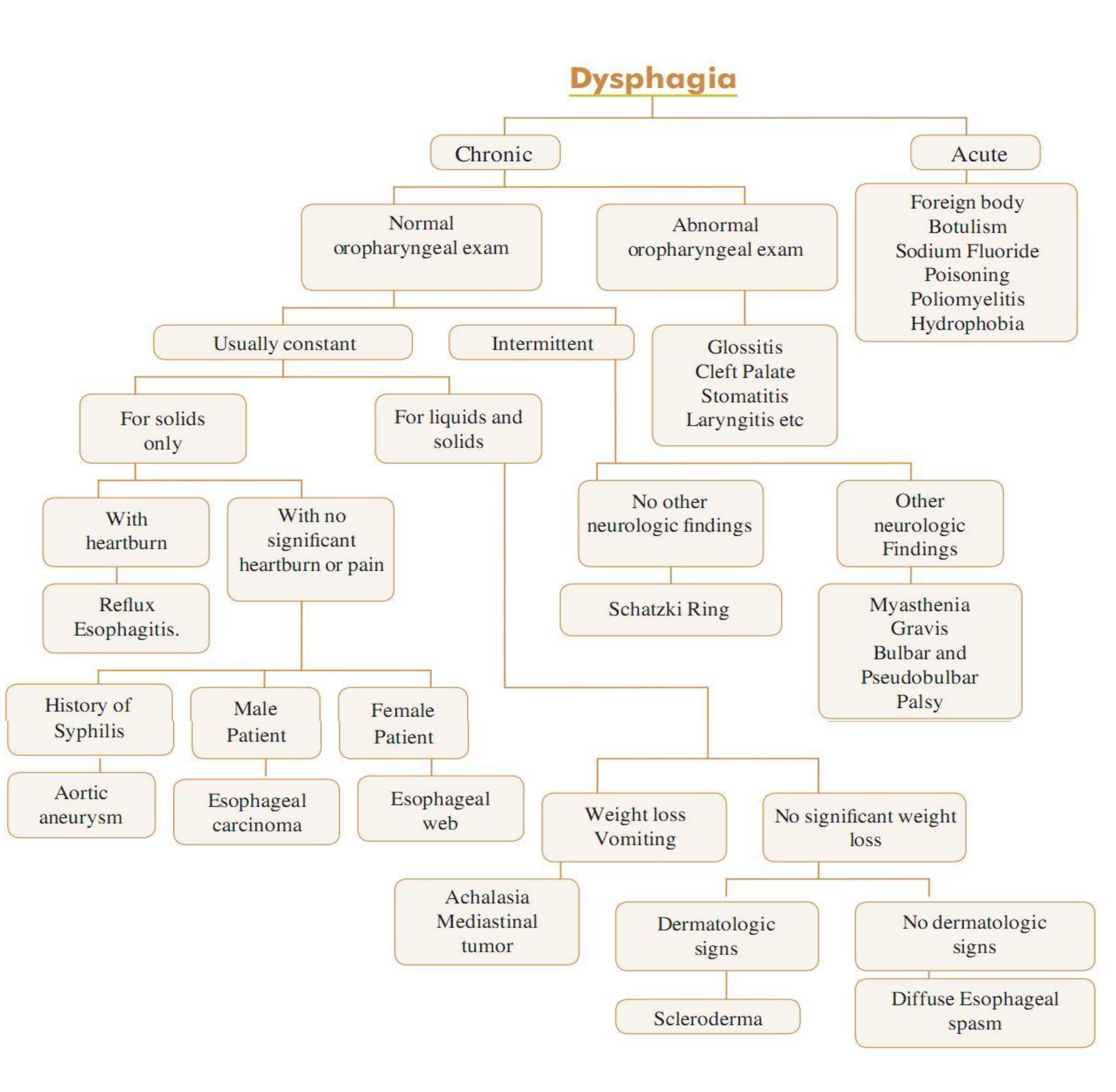

Melena
False
Iron
Charcoal
Beet Root
True
No history
of drug ingestion
History of Drug
Ingestion
Alcoholism
Aspirin
Anticoagulants
Caffeine
Reserpine
etc.
With
Hematemesis
Esophageal Varices
Peptic Ulcer
Gastritis etc.
(see Hematemesis)
Without
Hematemesis
With Abdominal
Pain
Without Significant
Abdominal Pain
Duodenal ulcer
Gastric Ulcer
Esophagitis
Mesenteric Embolism or
Thrombosis
Meckel's
Diverticulum
Gastric malignancy
Blood Dyscrasias
Typhoid Fever
Carcinoma of the
Ampulla of Vater
Hereditary Telangiectasia

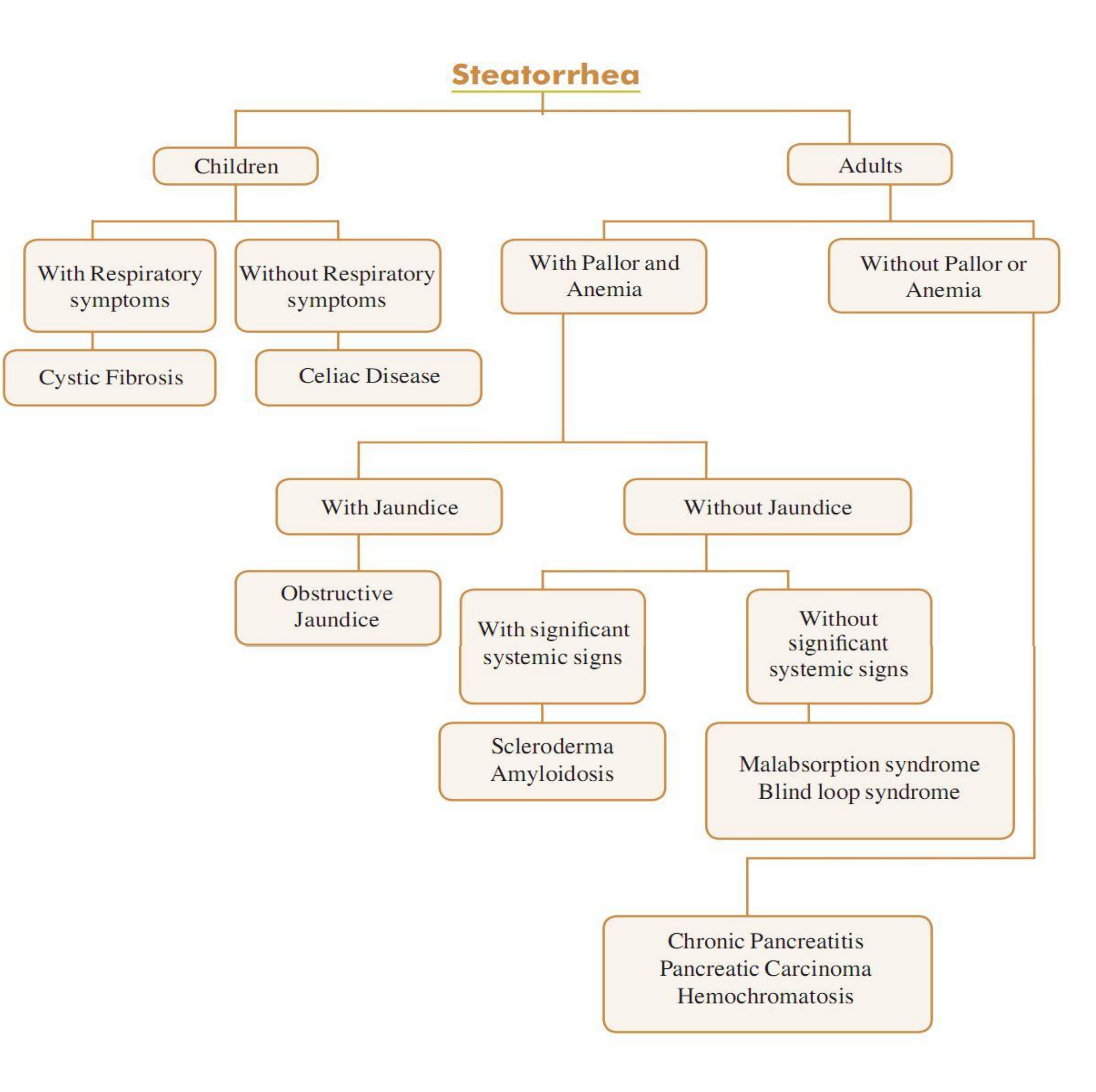

Acute Diarrhea
Blood in Stool
No Blood in Stool
Fever
No Fever
Severe Vomiting
Little or no Vomiting
Salmonella
Shigella
Campylobacter
Jejuni
Ulcerative Colitis
Amoebic
Dysentery
Giardiasis
Pseudomembraneous Colitis
Toxic staphylococcal
Gastroenteritis
Traveler's diarrhea
Contaminated food
Viral gastroenteritis


Chronic Diarrhea
Blood in Stool
No Blood in Stool
Mucus
No Mucus
Large amount of Mucus
No / small amount of Mucus
Ulcerative Colitis
Crohn's Disease
Pseudo-Membranous Colitis
Carcinoma
Diverticulitis
Amoebiasis
Zollinger-Ellison Syndrome
Irritable Bowel Syndrome
Signs of systemic disease
No signs of systemic disease
Thyrotoxicosis
Hyperpara-thyroidism
Fibrocystic disease
Pellagra
Carcinoid syndrome
Addison's disease
Amyloidosis
Diabetes mellitus
Pernicious anemia
Large-volume diarrhea
Persists during fasting
Stops during fasting
Zollinger-Ellison syndrome
Vasoactive Intestinal Polypeptide Tumor
Toxigenic bacteria
Giardiasis
Surreptitious drug ingestion
Lactase deficiency
Steatorrhea
Other disaccharidase Deficiency

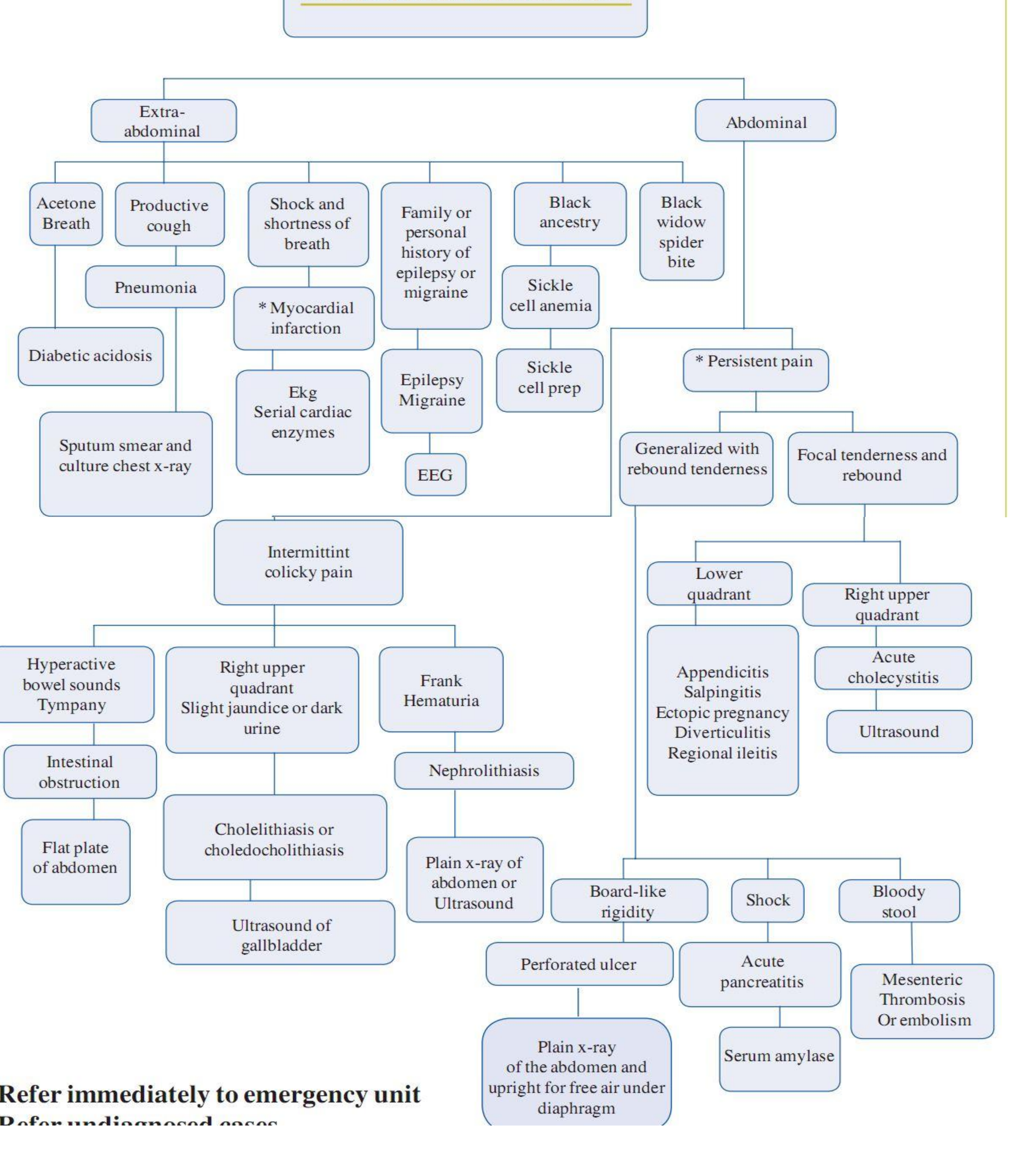

Nausea and Vomiting
No history of drug ingestion
History of drug ingestion
Alcoholism
Digitalis intoxication
Aspirin
Nonsteroidal
Anti-inflammatory drugs
Antihypertensives
etc.
Fever
No Fever
Abdominal pain
No abdominal pain
Acute hepatitis
Cholecystitis
Acute appendicitis
Peritonitis
Pyelonephritis
Gastroenteritis
Streptococcal
Pharyngitis in children
Meningitis
Otitis media
Any febrile illness
Constant or nearly constant abdominal pain
Intermittent Abdominal pain
No abdominal pain
Renal colic
Biliary colic
Vertigo
No vertigo
Meniere's disease
Labyrinthitis
Pyloric or intestinal
Obstruction
Perinephric Abscess
Acute cholecystitis
Pregnancy
Pancreatic neoplasm
Acute and chronic
pancreatitis
Gastritis
Ulcer
Acute appendicitis
Diabetic acidosis
Headache
No headache
Migraine
Concussion
Cerebaal tumor or
Other mass
Food poisoning
Pregnancy
Hypercalcemia
Uremia
Pernicious anemia
Angioneurotic edema
Malingering

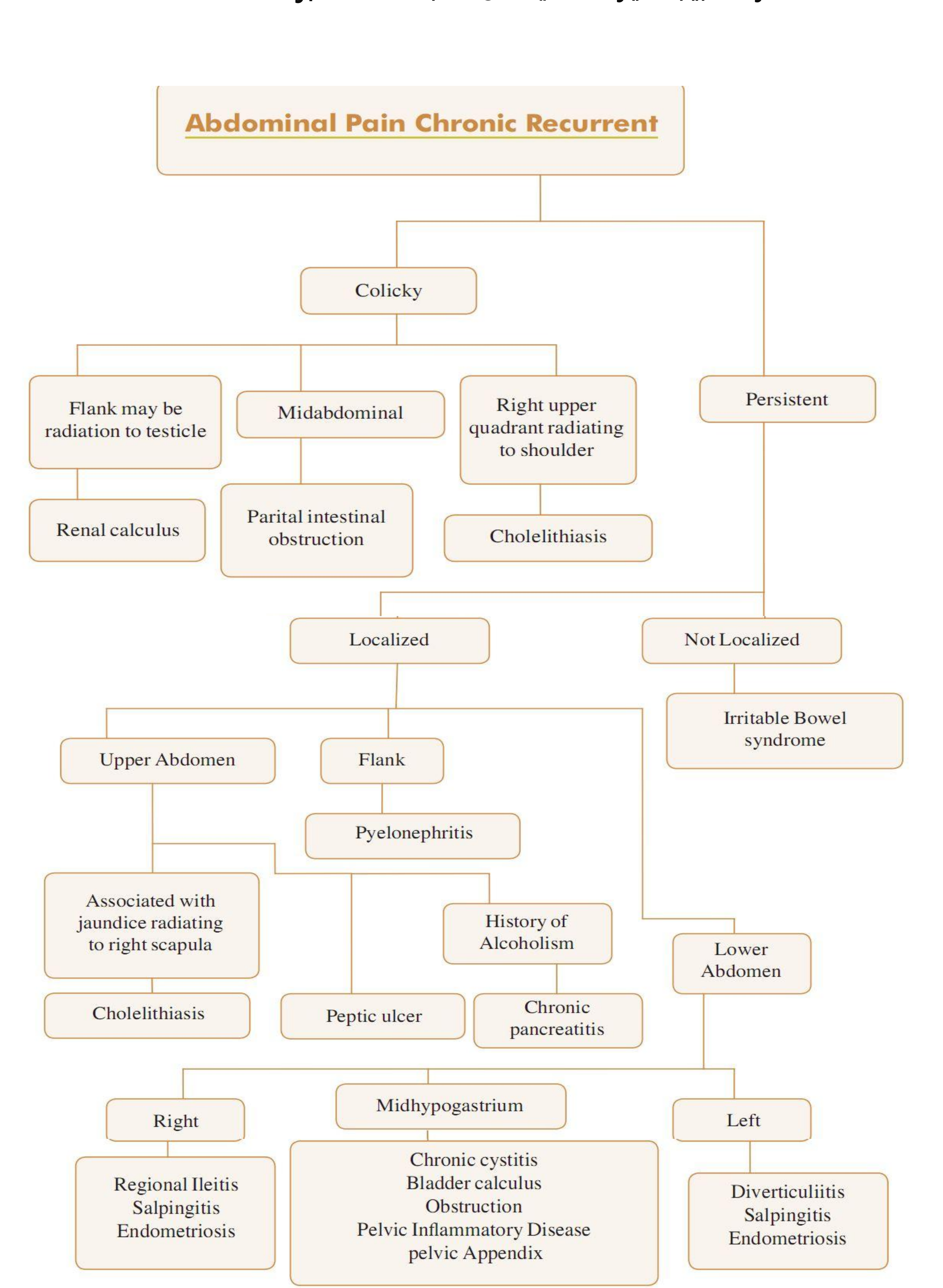

Constipation
Abdominal Pain or Vomiting
No Abdominal Pain or Vomiting
Complete Intestinal Obstruction
Incomplete Intestinal Obstruction
Blood in the Stool
No Blood in the Stool
Drugs
Diet
Habit
Functional
Irritable Bowel Syndrome
Painful Defecation
Painless Defecation
Hemorrhoids
anal Fissure
Colon Carcinoma
Diverticulitis

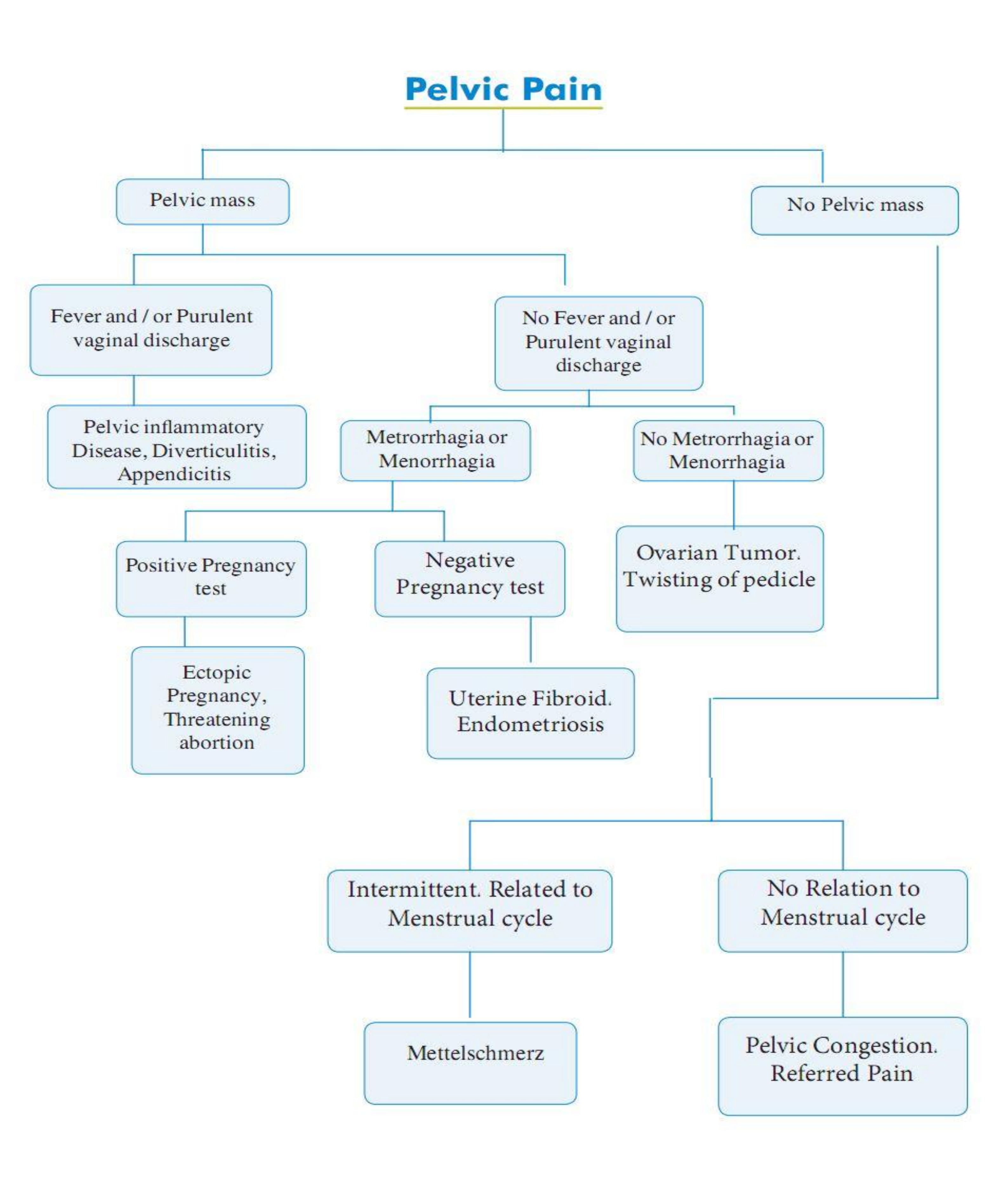

Dysphagia
Chronic
Acute
Foreign body
Botulism
Sodium Fluoride Poisoning
Poliomyelitis
Hydrophobia
Normal oropharyngeal exam
Abnormal oropharyngeal exam
Glossitis
Cleft Palate
Stomatitis
Laryngitis etc
Usually constant
Intermittent
For solids only
For liquids and solids
No other neurologic findings
Other neurologic Findings
Schatzki Ring
Myasthenia Gravis
Bulbar and Pseudobulbar Palsy
With heartburn
Reflux Esophagitis.
With no significant heartburn or pain
History of Syphilis
Aortic aneurysm
Male Patient
Esophageal carcinoma
Female Patient
Esophageal web
Weight loss Vomiting
Achalasia
Mediastinal tumor
No significant weight loss
Dermatologic signs
Scleroderma
No dermatologic signs
Diffuse Esophageal spasm


Steatorrhea
Children
Adults
With Respiratory symptoms
Without Respiratory symptoms
With Pallor and Anemia
Without Pallor or Anemia
Cystic Fibrosis
Celiac Disease
With Jaundice
Without Jaundice
Obstructive Jaundice
With significant systemic signs
Without significant systemic signs
Scleroderma
Amyloidosis
Malabsorption syndrome
Blind loop syndrome
Chronic Pancreatitis
Pancreatic Carcinoma
Hemochromatosis

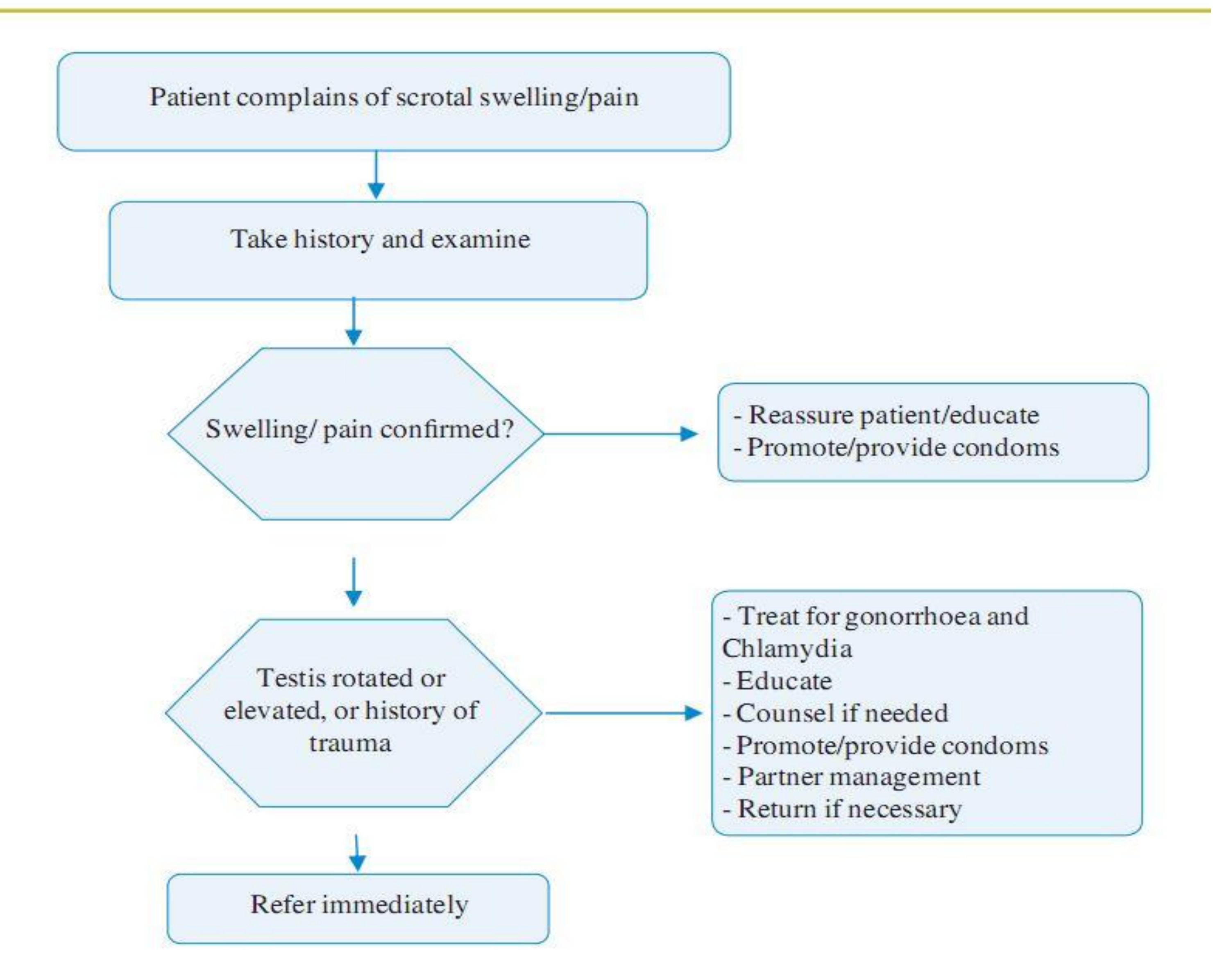
Acute Abdominal Pain

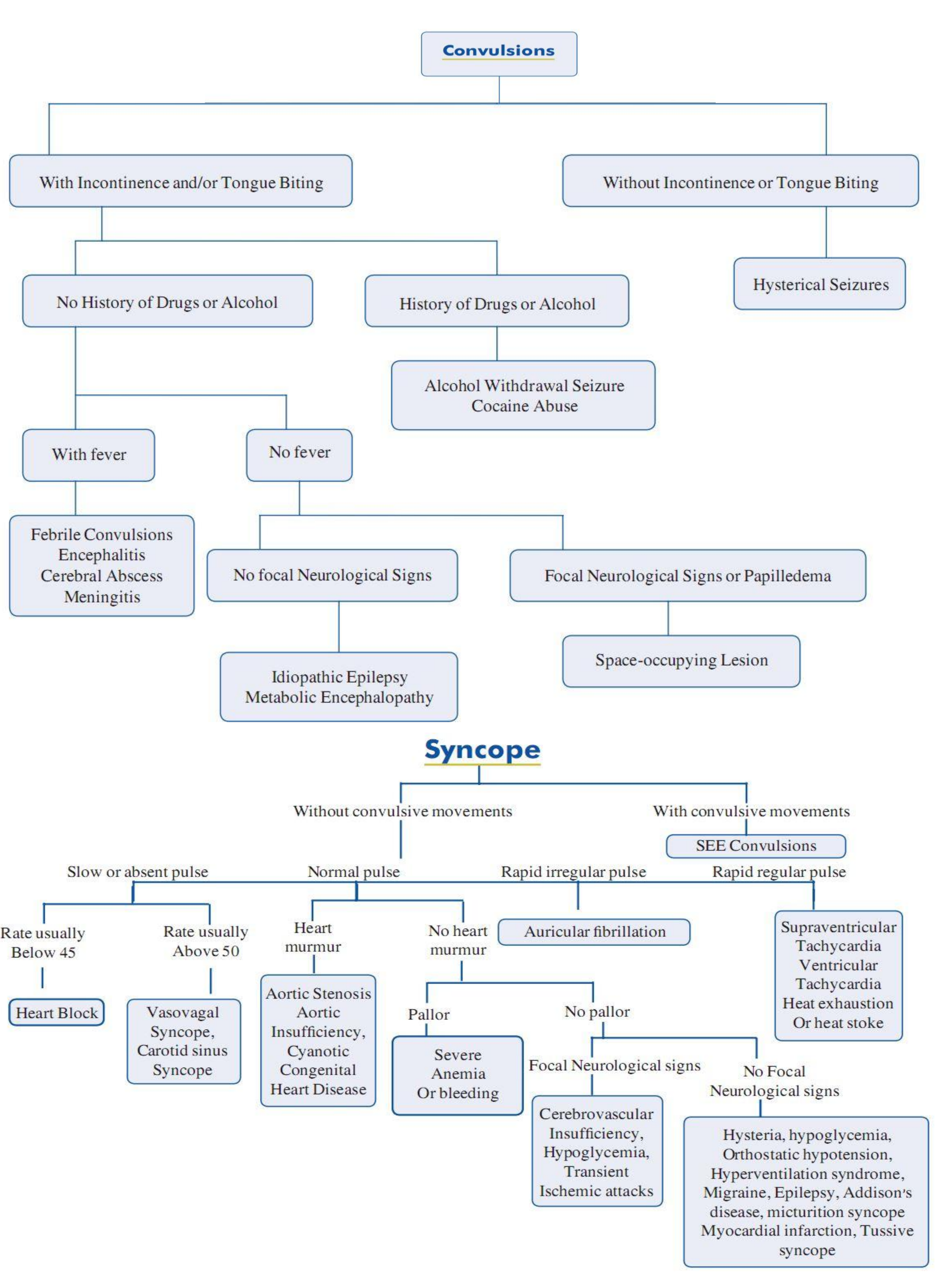


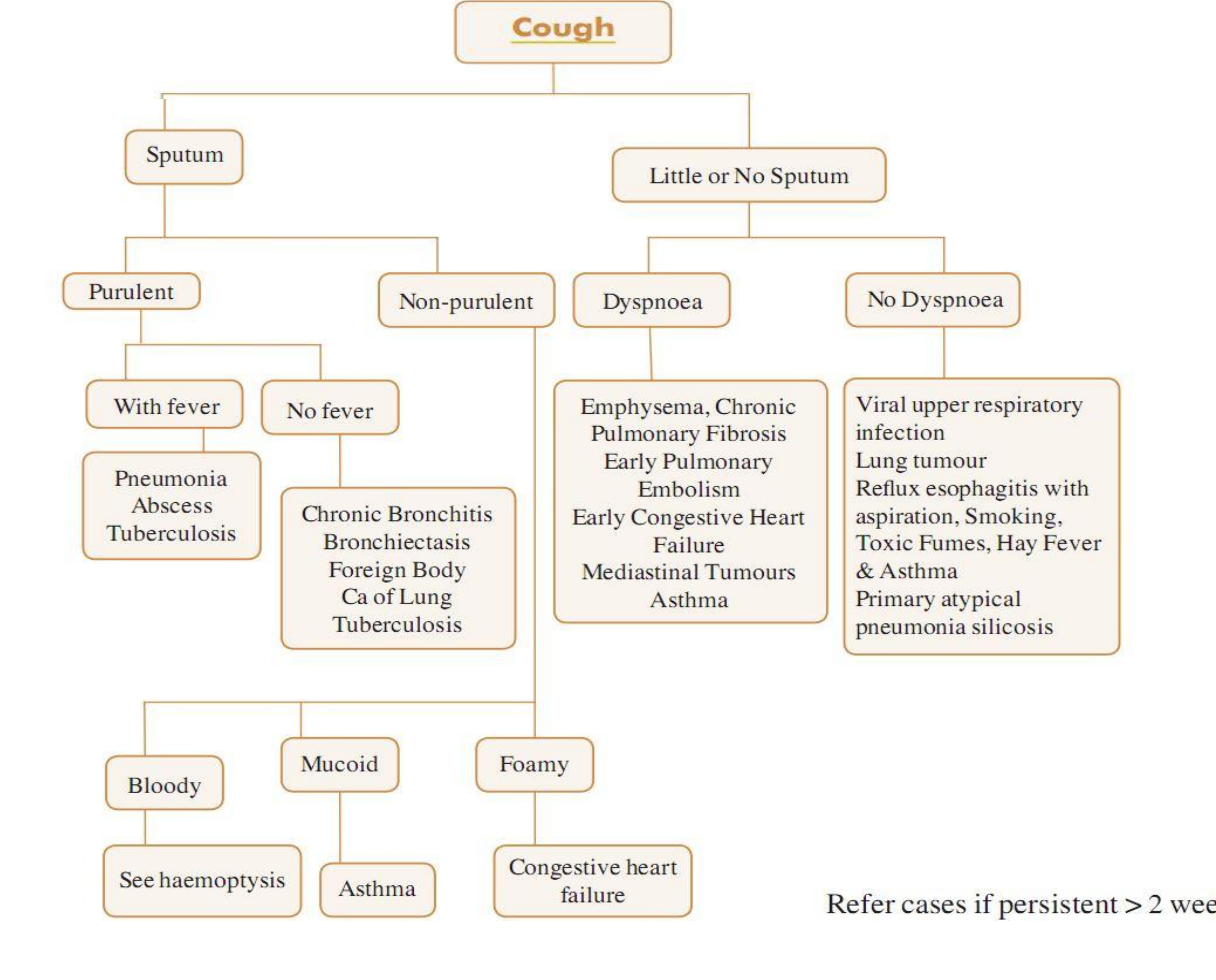

Abdominal Pain Chronic Recurrent
Colicky
Persistent
Flank may be radiation to testicle
Midabdominal
Right upper quadrant radiating to shoulder
Renal calculus
Parital intestinal obstruction
Cholelithiasis
Localized
Not Localized
Irritable Bowel syndrome
Upper Abdomen
Flank
Pyelonephritis
Associated with jaundice radiating to right scapula
History of Alcoholism
Lower Abdomen
Cholelithiasis
Peptic ulcer
Chronic pancreatitis
Right
Midhypogastrium
Left
Regional Ileitis
Salpingitis
Endometriosis
Chronic cystitis
Bladder calculus
Obstruction
Pelvic Inflammatory Disease
pelvic Appendix
Diverticuliitis
Salpingitis
Endometriosis

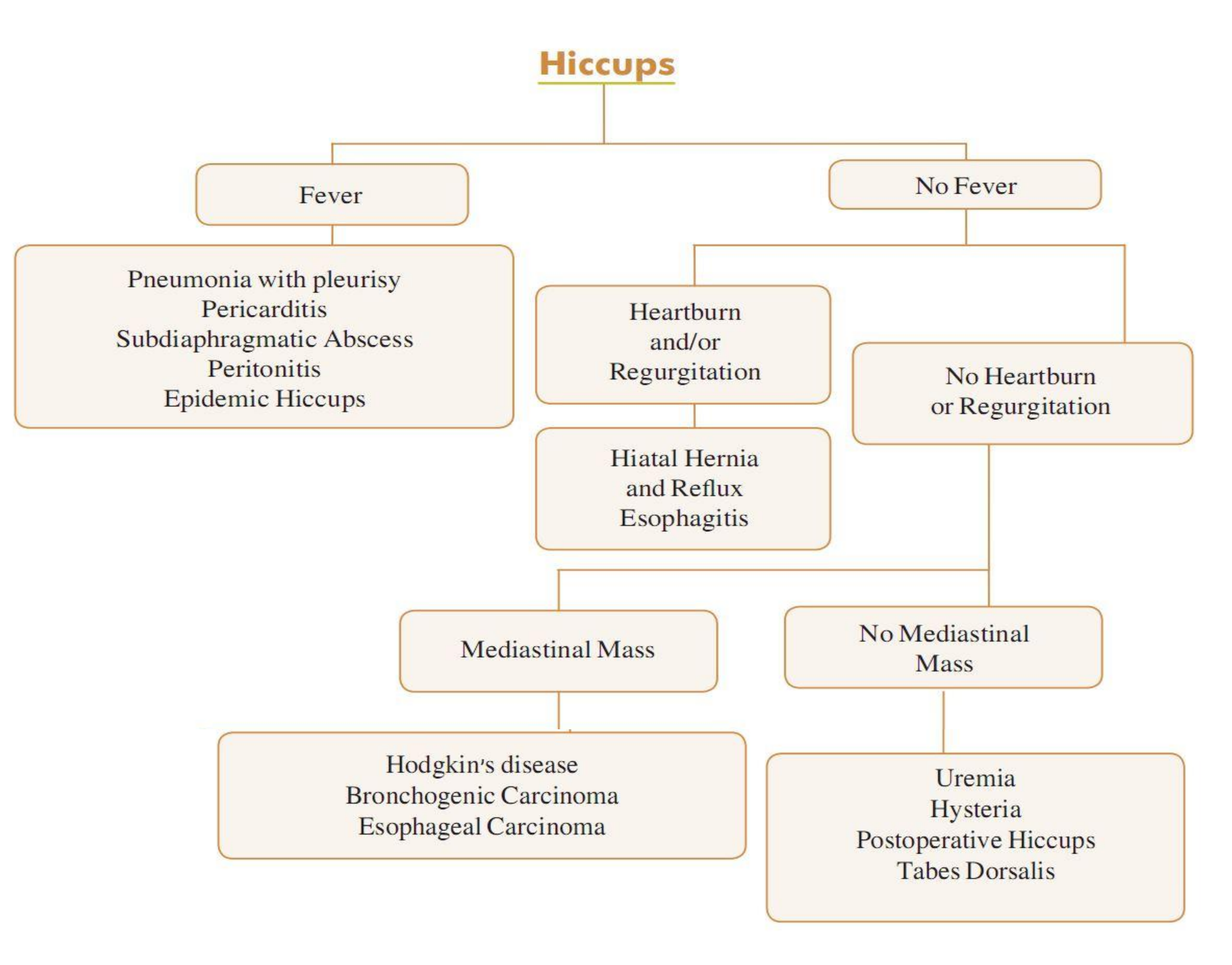

Pelvic Pain
Pelvic mass
No Pelvic mass
Fever and / or Purulent vaginal discharge
No Fever and / or Purulent vaginal discharge
Pelvic inflammatory Disease, Diverticulitis, Appendicitis
Metrorrhagia or Menorrhagia
No Metrorrhagia or Menorrhagia
Positive Pregnancy test
Negative Pregnancy test
Ovarian Tumor. Twisting of pedicle
Ectopic Pregnancy, Threatening abortion
Uterine Fibroid. Endometriosis
Intermittent. Related to Menstrual cycle
No Relation to Menstrual cycle
Mettelschmerz
Pelvic Congestion. Referred Pain

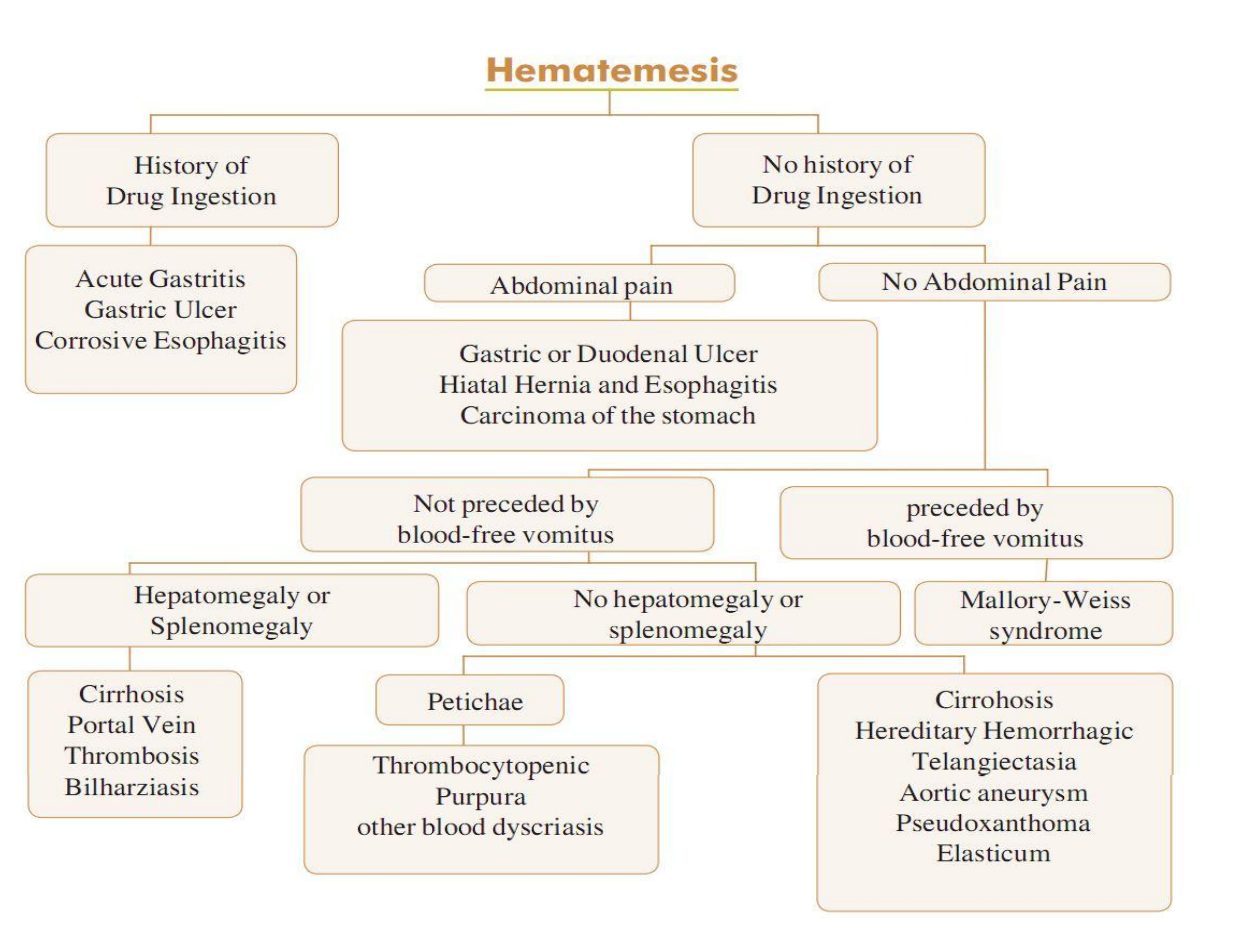
Scrotal Swelling

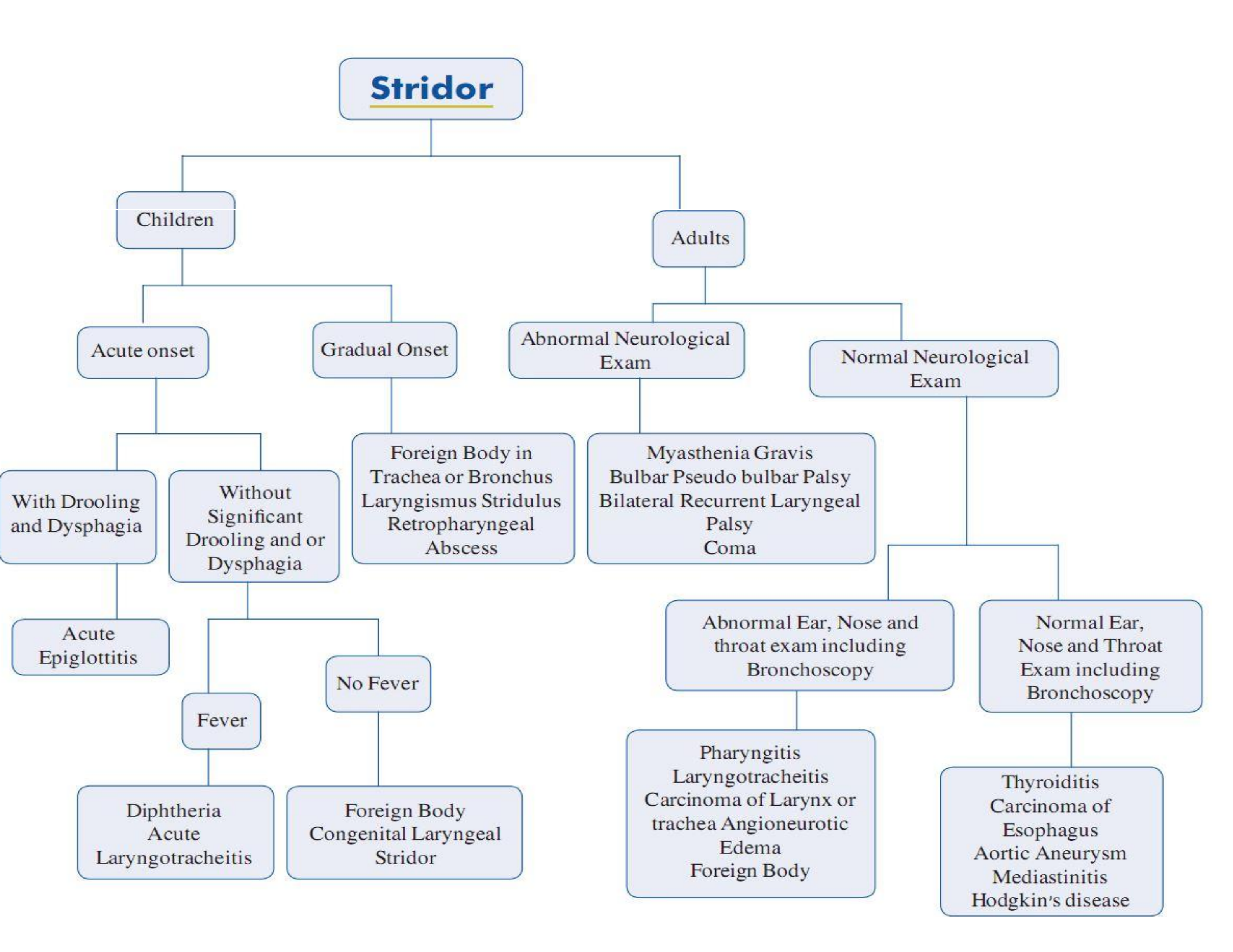


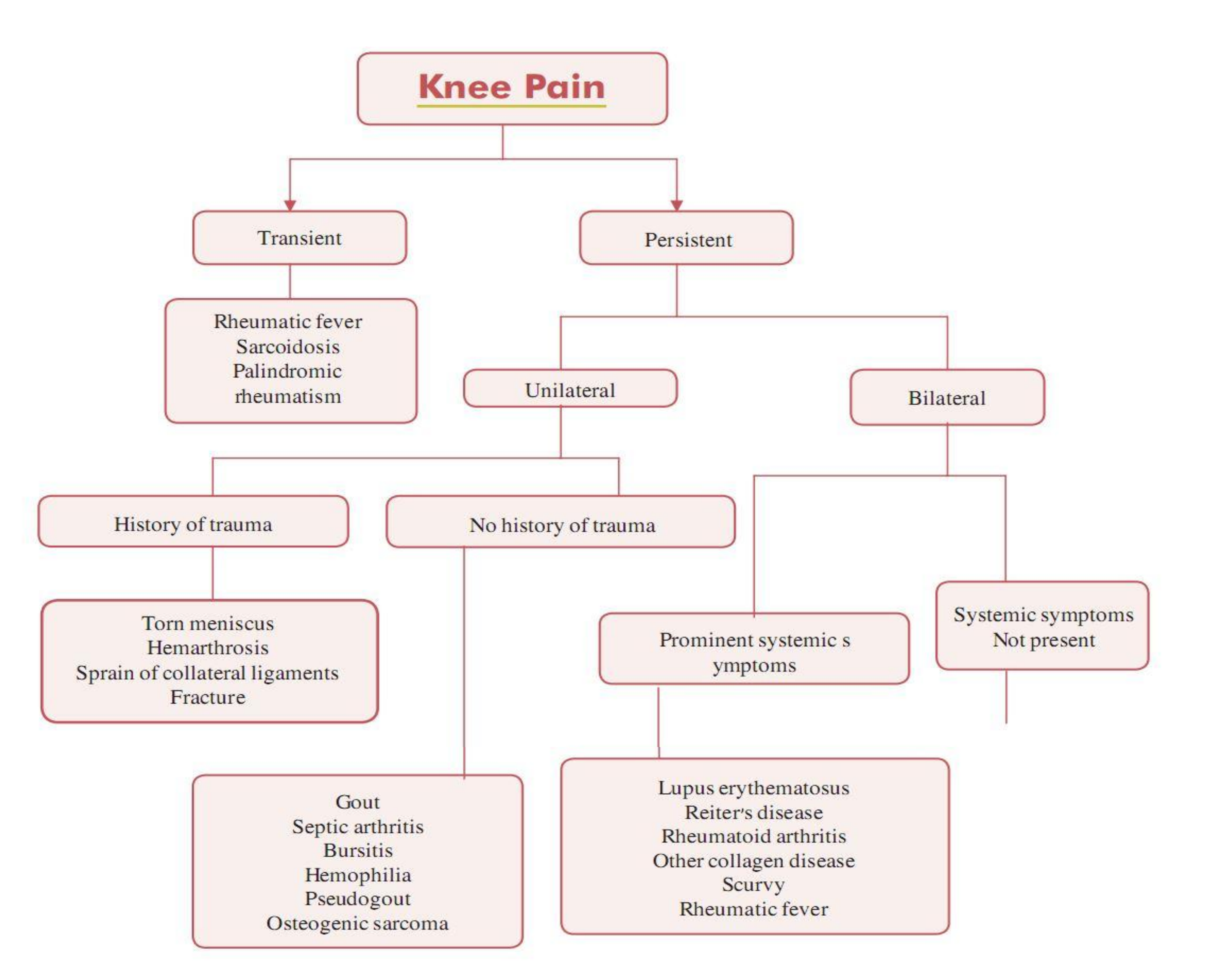

Convulsions
With Incontinence and/or Tongue Biting
Without Incontinence or Tongue Biting
Hysterical Seizures
No History of Drugs or Alcohol
History of Drugs or Alcohol
Alcohol Withdrawal Seizure
Cocaine Abuse
With fever
No fever
Febrile Convulsions
Encephalitis
Cerebral Abscess
Meningitis
No focal Neurological Signs
Focal Neurological Signs or Papilledema
Idiopathic Epilepsy
Metabolic Encephalopathy
Space-occupying Lesion
Syncope
Without convulsive movements
With convulsive movements
SEE Convulsions
Slow or absent pulse
Normal pulse
Rapid irregular pulse
Rapid regular pulse
Rate usually Below 45
Rate usually Above 50
Heart murmur
No heart murmur
Auricular fibrillation
Supraventricular Tachycardia
Ventricular Tachycardia
Heat exhaustion
Or heat stoke
Heart Block
Vasovagal Syncope,
Carotid sinus Syncope
Aortic Stenosis
Aortic Insufficiency,
Cyanotic Congenital Heart Disease
Pallor
No pallor
Severe Anemia
Or bleeding
Focal Neurological signs
No Focal Neurological signs
Cerebrovascular Insufficiency,
Hypoglycemia,
Transient Ischemic attacks
Hysteria, hypoglycemia,
Orthostatic hypotension,
Hyperventilation syndrome,
Migraine, Epilepsy, Addison's disease, micturition syncope
Myocardial infarction, Tussive syncope

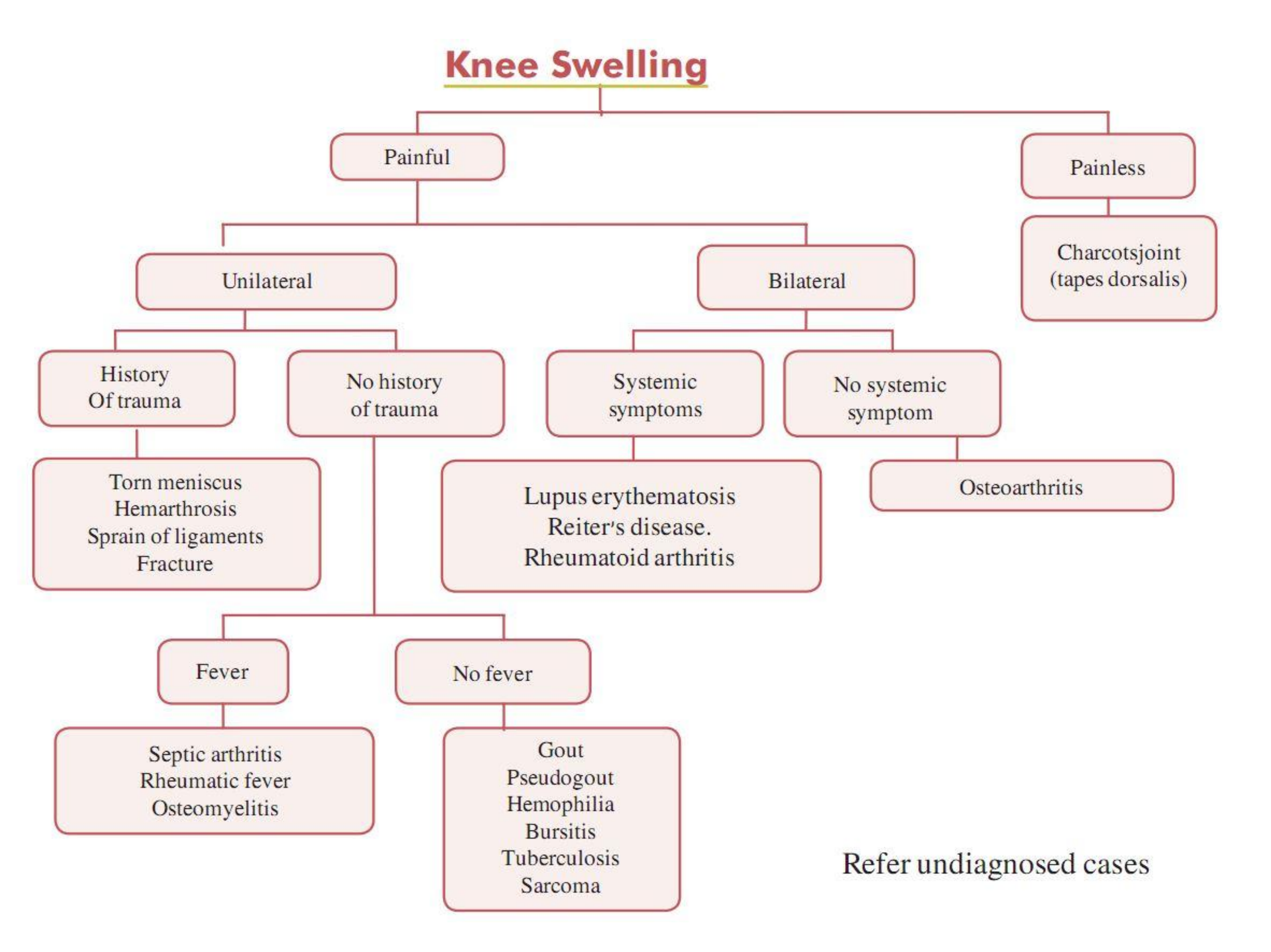

Cough
Sputum
Little or No Sputum
Purulent
Non-purulent
Dyspnoea
No Dyspnoea
With fever
No fever
Pneumonia
Abscess
Tuberculosis
Chronic Bronchitis
Bronchiectasis
Foreign Body
Ca of Lung
Tuberculosis
Emphysema, Chronic
Pulmonary Fibrosis
Early Pulmonary
Embolism
Early Congestive Heart
Failure
Mediastinal Tumours
Asthma
Viral upper respiratory
infection
Lung tumour
Reflux esophagitis with
aspiration, Smoking,
Toxic Fumes, Hay Fever
& Asthma
Primary atypical
pneumonia silicosis
Bloody
Mucoid
Foamy
See haemoptysis
Asthma
Congestive heart
failure
Refer cases if persistent > 2 wee

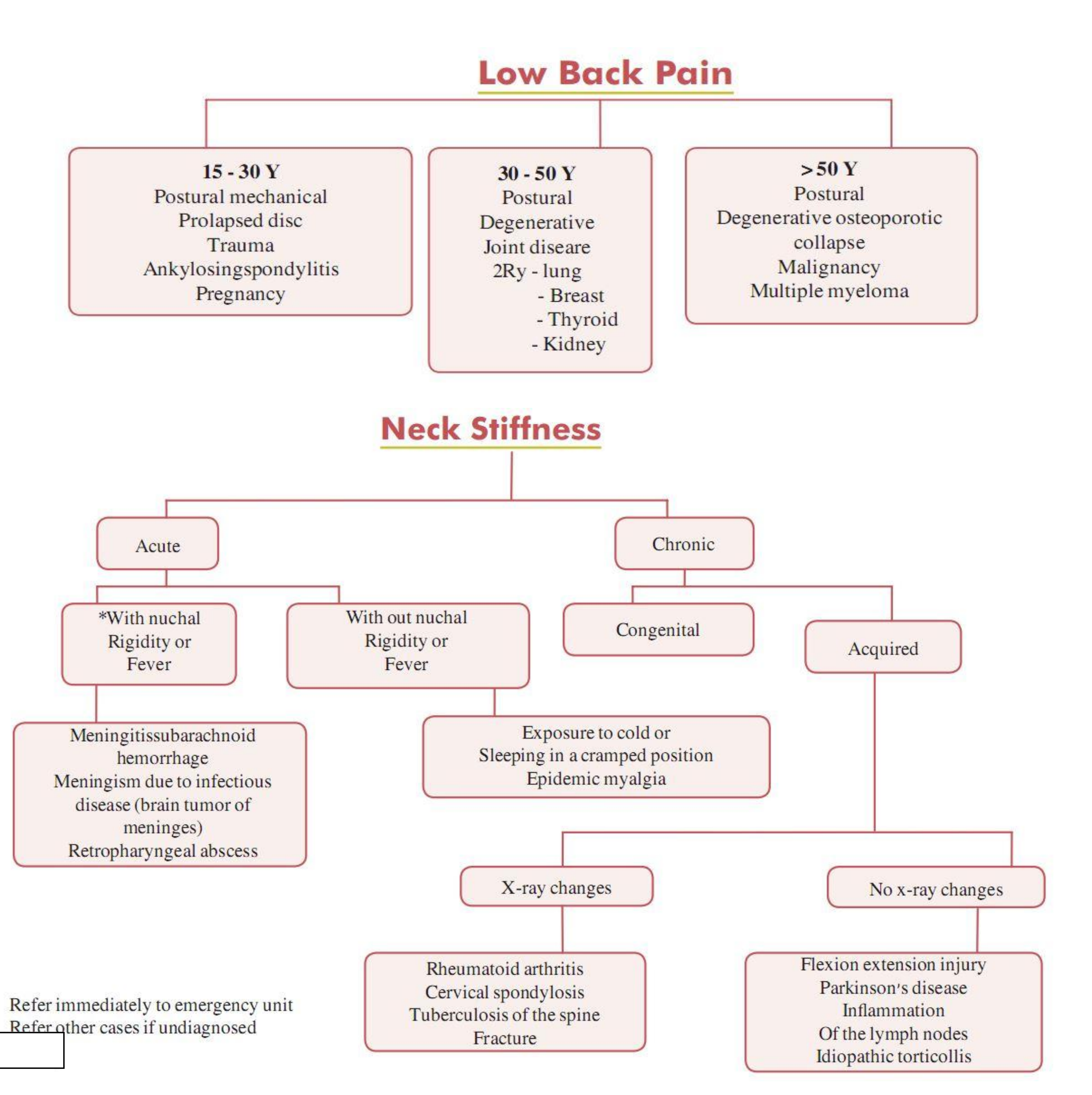

Hiccups
Fever
No Fever
Pneumonia with pleurisy
Pericarditis
Subdiaphragmatic Abscess
Peritonitis
Epidemic Hiccups
Heartburn
and/or
Regurgitation
No Heartburn
or Regurgitation
Hiatal Hernia
and Reflux
Esophagitis
Mediastinal Mass
No Mediastinal
Mass
Hodgkin's disease
Bronchogenic Carcinoma
Esophageal Carcinoma
Uremia
Hysteria
Postoperative Hiccups
Tabes Dorsalis

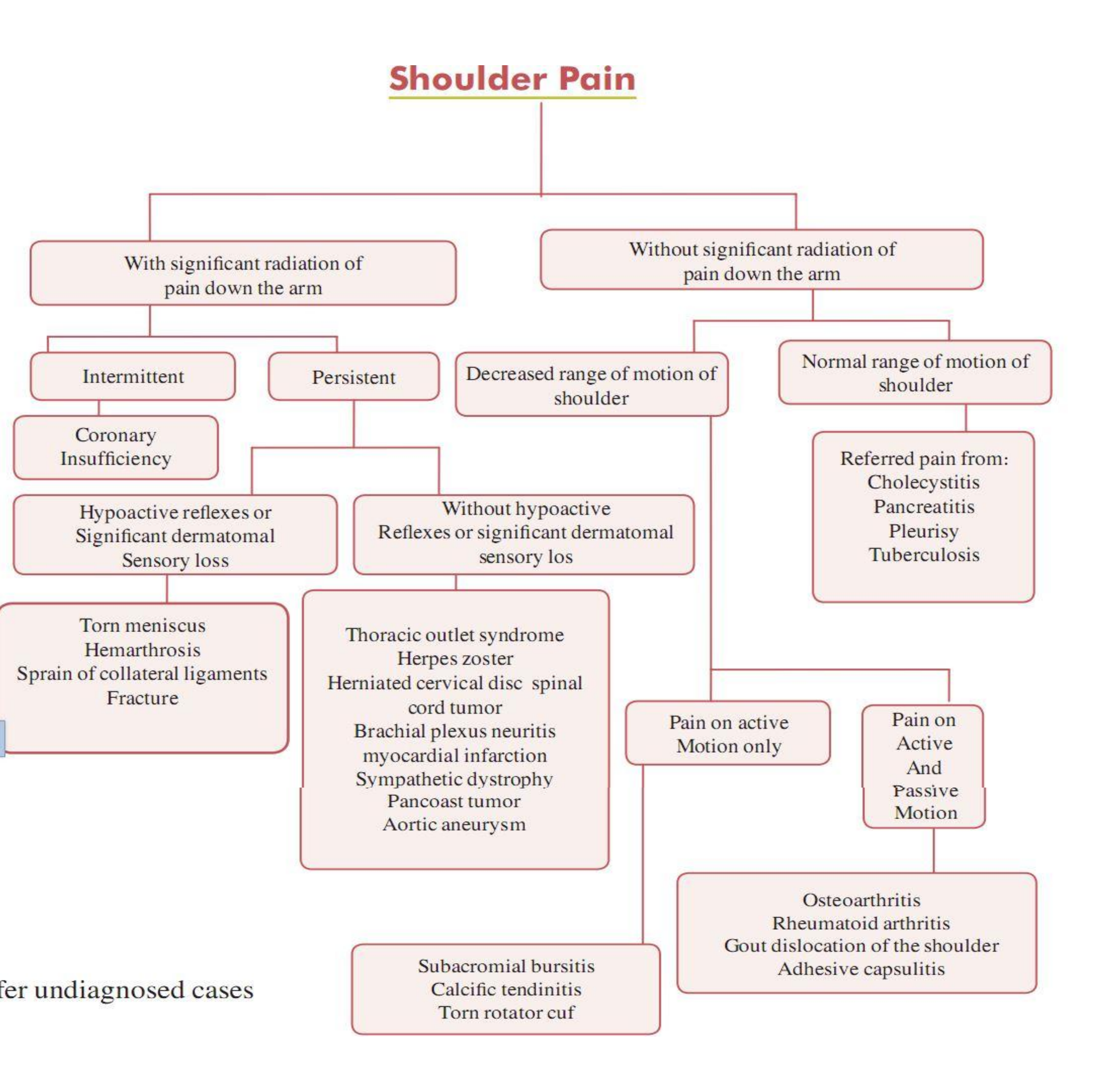

Hematemesis
History of Drug Ingestion
Acute Gastritis
Gastric Ulcer
Corrosive Esophagitis
No history of Drug Ingestion
Abdominal pain
No Abdominal Pain
Gastric or Duodenal Ulcer
Hiatal Hernia and Esophagitis
Carcinoma of the stomach
Not preceded by blood-free vomitus
preceded by blood-free vomitus
Hepatomegaly or Splenomegaly
No hepatomegaly or splenomegaly
Mallory-Weiss syndrome
Cirrhosis
Portal Vein Thrombosis
Bilharziasis
Petichae
Thrombocytopenic Purpura
other blood dyscriasis
Cirrohosis
Hereditary Hemorrhagic Telangiectasia
Aortic aneurysm
Pseudoxanthoma Elasticum

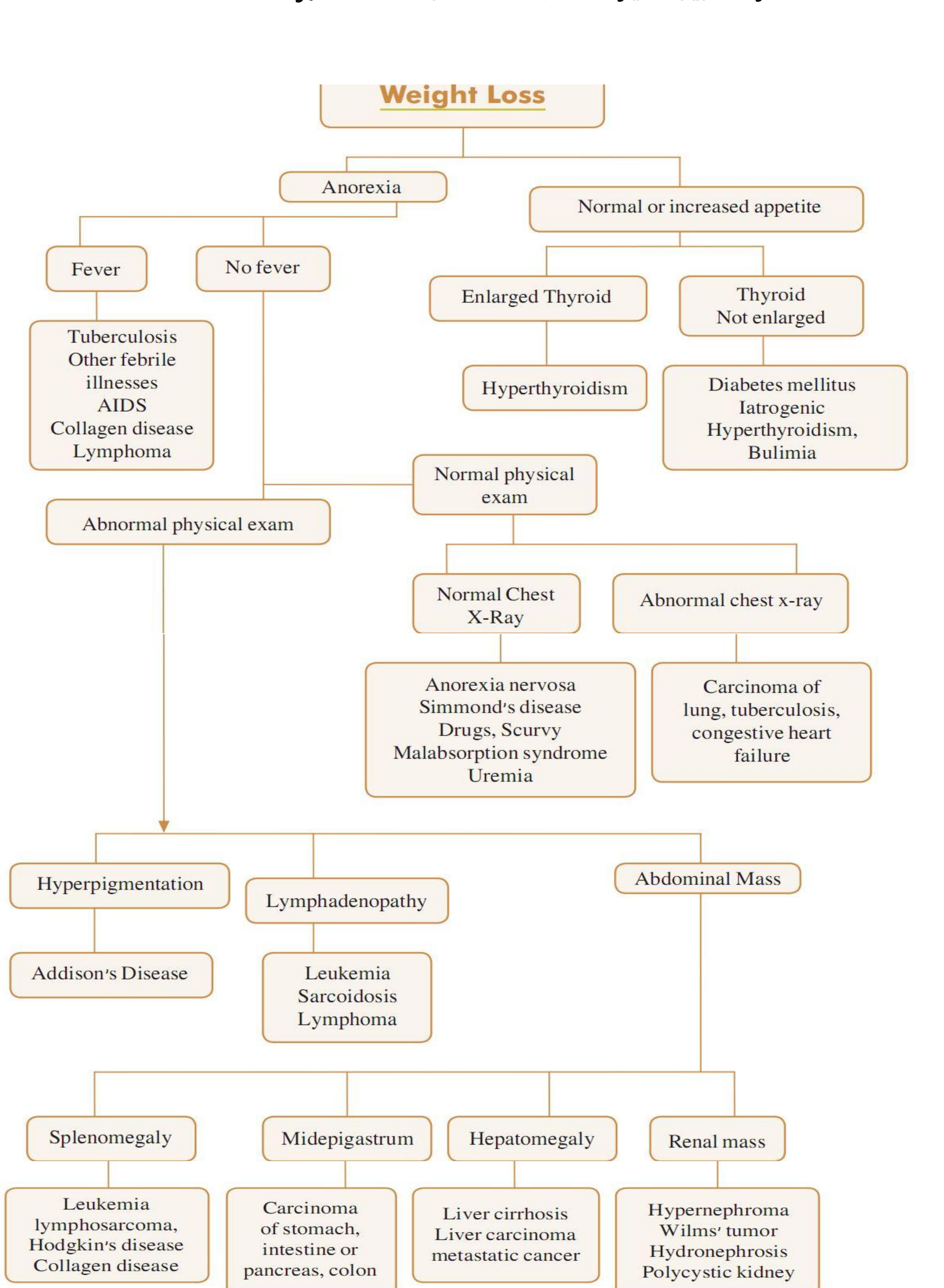

Stridor
Children
Adults
Acute onset
Gradual Onset
Abnormal Neurological Exam
Normal Neurological Exam
With Drooling and Dysphagia
Without Significant Drooling and or Dysphagia
Foreign Body in Trachea or Bronchus Laryngismus Stridulus Retropharyngeal Abscess
Myasthenia Gravis Bulbar Pseudo bulbar Palsy Bilateral Recurrent Laryngeal Palsy Coma
Acute Epiglottitis
Fever
No Fever
Abnormal Ear, Nose and throat exam including Bronchoscopy
Normal Ear, Nose and Throat Exam including Bronchoscopy
Diphtheria Acute Laryngotracheitis
Foreign Body Congenital Laryngeal Stridor
Pharyngitis Laryngotracheitis Carcinoma of Larynx or trachea Angioneurotic Edema Foreign Body
Thyroiditis Carcinoma of Esophagus Aortic Aneurysm Mediastinitis Hodgkin's disease


Knee Pain
Transient
Persistent
Rheumatic fever
Sarcoidosis
Palindromic
rheumatism
Unilateral
Bilateral
History of trauma
No history of trauma
Torn meniscus
Hemarthrosis
Sprain of collateral ligaments
Fracture
Prominent systemic s
ymptoms
Systemic symptoms
Not present
Gout
Septic arthritis
Bursitis
Hemophilia
Pseudogout
Osteogenic sarcoma
Lupus erythematosus
Reiter's disease
Rheumatoid arthritis
Other collagen disease
Scurvy
Rheumatic fever


Knee Swelling
Painful
Painless
Charcotsjoint
(tapes dorsalis)
Unilateral
Bilateral
History
Of trauma
No history
of trauma
Systemic
symptoms
No systemic
symptom
Torn meniscus
Hemarthrosis
Sprain of ligaments
Fracture
Lupus erythematosis
Reiter's disease.
Rheumatoid arthritis
Osteoarthritis
Fever
No fever
Septic arthritis
Rheumatic fever
Osteomyelitis
Gout
Pseudogout
Hemophilia
Bursitis
Tuberculosis
Sarcoma
Refer undiagnosed cases


Low Back Pain
15 - 30 Y
Postural mechanical
Prolapsed disc
Trauma
Ankylosingspondylitis
Pregnancy
30 - 50 Y
Postural
Degenerative
Joint diseare
2Ry - lung
- Breast
- Thyroid
- Kidney
> 50 Y
Postural
Degenerative osteoporotic collapse
Malignancy
Multiple myeloma
Neck Stiffness
Acute
Chronic
*With nuchal Rigidity or Fever
With out nuchal Rigidity or Fever
Congenital
Acquired
Meningitissubarachnoid hemorrhage
Meningism due to infectious disease (brain tumor of meninges)
Retropharyngeal abscess
Exposure to cold or
Sleeping in a cramped position
Epidemic myalgia
X-ray changes
No x-ray changes
Rheumatoid arthritis
Cervical spondylosis
Tuberculosis of the spine
Fracture
Flexion extension injury
Parkinson's disease
Inflammation
Of the lymph nodes
Idiopathic torticollis
Refer immediately to emergency unit
Refer other cases if undiagnosed


Shoulder Pain
With significant radiation of pain down the arm
Without significant radiation of pain down the arm
Intermittent
Persistent
Decreased range of motion of shoulder
Normal range of motion of shoulder
Coronary Insufficiency
Referred pain from: Cholecystitis Pancreatitis Pleurisy Tuberculosis
Hypoactive reflexes or Significant dermatomal Sensory loss
Without hypoactive Reflexes or significant dermatomal sensory los
Torn meniscus Hemarthrosis Sprain of collateral ligaments Fracture
Thoracic outlet syndrome Herpes zoster Herniated cervical disc spinal cord tumor Brachial plexus neuritis myocardial infarction Sympathetic dystrophy Pancoast tumor Aortic aneurysm
Pain on active Motion only
Pain on Active And Passive Motion
Osteoarthritis Rheumatoid arthritis Gout dislocation of the shoulder Adhesive capsulitis
Subacromial bursitis Calcific tendinitis Torn rotator cuf
fer undiagnosed cases


Weight Loss
Anorexia
Normal or increased appetite
Fever
No fever
Tuberculosis
Other febrile illnesses
AIDS
Collagen disease
Lymphoma
Enlarged Thyroid
Thyroid
Not enlarged
Hyperthyroidism
Diabetes mellitus
Iatrogenic
Hyperthyroidism,
Bulimia
Normal physical exam
Abnormal physical exam
Normal Chest X-Ray
Abnormal chest x-ray
Anorexia nervosa
Simmond's disease
Drugs, Scurvy
Malabsorption syndrome
Uremia
Carcinoma of lung, tuberculosis, congestive heart failure
Hyperpigmentation
Lymphadenopathy
Abdominal Mass
Addison's Disease
Leukemia
Sarcoidosis
Lymphoma
Splenomegaly
Midepigastrum
Hepatomegaly
Renal mass
Leukemia
lymphosarcoma,
Hodgkin's disease
Collagen disease
Carcinoma
of stomach,
intestine or
pancreas, colon
Liver cirrhosis
Liver carcinoma
metastatic cancer
Hypernephroma
Wilms' tumor
Hydronephrosis
Polycystic kidney

Hypertension

Background

Hypertension is one of the most common worldwide diseases afflicting humans and is a major risk factor for stroke, myocardial infarction, vascular disease, and chronic kidney disease. Due to the associated morbidity and mortality and cost to society, preventing and treating hypertension is an important public health challenge.

Hypertension is the most important modifiable risk factor for coronary heart disease (the leading cause of death in North America), stroke (the third leading cause), congestive heart failure, end-stage renal disease, and peripheral vascular disease. Therefore, health care professionals must not only identify and treat patients with hypertension but also promote a healthy lifestyle and preventive strategies to decrease the prevalence of hypertension in the general population.

Definition and classification

Based on recommendations of the JNC 7, the classification of BP (expressed in mm Hg) for adults aged 18 years or older is as follows[1]:

- **Normal**: systolic lower than 120 mm Hg, diastolic lower than 80 mm Hg
- **Prehypertension**: systolic 120-139 mm Hg, diastolic 80-89 mm Hg
- **Stage 1**: systolic 140-159 mm Hg, diastolic 90-99 mm Hg
- **Stage 2**: systolic 160 mm Hg or greater, diastolic 100 mm Hg or greater

The classification above is based on the average of 2 or more readings taken at each of 2 or more visits after initial screening.

Prehypertension, a new category designated in the JNC 7 report, emphasizes that patients with prehypertension are at risk for progression to hypertension and that lifestyle modifications are important preventive strategies.

Hypertension may be categorized as either essential or secondary.

Primary (essential) hypertension is diagnosed in the absence of an identifiable secondary cause. Approximately 90-95% of adults with hypertension have primary hypertension, whereas secondary hypertension accounts for around 5-10% of the cases.

Especially severe cases of hypertension, or **hypertensive crises**, are defined as a BP of more than 180/120 mm Hg and may be further categorized as hypertensive emergencies or urgencies. Hypertensive emergencies are characterized by evidence of impending or progressive target organ dysfunction, whereas hypertensive urgencies are those situations without progressive target organ dysfunction.

In hypertensive emergencies, the BP should be aggressively lowered within minutes to an hour by no more than 25%, and then lowered to 160/100-110 mm Hg within the next 2-6 hours.

Acute end-organ damage in the setting of a hypertensive emergency may include the following:

- Neurologic: hypertensive encephalopathy, cerebral vascular accident/cerebral infarction, subarachnoid hemorrhage, intracranial hemorrhage
- Cardiovascular: myocardial ischemia/infarction, acute left ventricular dysfunction, acute pulmonary edema, aortic dissection, unstable angina pectoris
- Other: acute renal failure/insufficiency, retinopathy, eclampsia, microangiopathic hemolytic anemia

Pathophysiology

The pathogenesis of essential hypertension is multifactorial and highly complex. Multiple factors modulate the blood pressure (BP) for adequate tissue perfusion and include humoral mediators, vascular reactivity, circulating blood volume, vascular caliber, blood viscosity, cardiac output, blood vessel elasticity, and neural stimulation. A possible pathogenesis of essential hypertension has been proposed in which multiple factors, including genetic predisposition, excess dietary salt intake, and adrenergic tone, may interact to produce hypertension. Although genetics appears to contribute to essential hypertension, the exact mechanism has not been established.

Etiology

Hypertension may be primary, which may develop as a result of environmental or genetic causes, or secondary, which has multiple etiologies, including renal, vascular, and endocrine causes. Primary or essential hypertension accounts for 90-95% of adult cases, and a small percentage of patients (2-10%) have a secondary cause. Hypertensive emergencies are most often precipitated by inadequate medication or poor compliance.

Causes of secondary hypertension

Renal causes (2.5-6%) of hypertension include the renal parenchymal diseases and renal vascular diseases, as follows:

- Polycystic kidney disease
- Chronic kidney disease
- Urinary tract obstruction
- Renin-producing tumor
- Liddle syndrome

Vascular causes include the following:

- Coarctation of aorta
- Vasculitis
- Collagen vascular disease

Endocrine causes exogenous or endogenous hormonal imbalances. Exogenous causes include - administration of steroids.

The most common form of secondary hypertension is a renal cause

Another common cause is endocrine: oral contraceptive use.

Activation of the renin-angiotensin-aldosterone system (RAAS) is the likely mechanism, because hepatic synthesis of angiotensinogen is induced by the estrogen

component of oral contraceptives. Approximately 5% of women taking oral contraceptives may develop hypertension, which abates within 6 months after discontinuation. The risk factors for oral contraceptive–associated hypertension include mild renal disease, familial history of essential hypertension, age older than 35 years, and obesity. It would be better to group oral contraceptives and steroids with drug-induced hypertension .

Exogenous administration of the other steroids used for therapeutic purposes also increases blood pressure (BP), especially in susceptible individuals, mainly by volume expansion.

Nonsteroidal anti-inflammatory drugs (NSAIDs) may also have adverse effects on BP. NSAIDs block both cyclooxygenase-1 (COX-1) and COX-2 enzymes. The inhibition of COX-2 can inhibit its natriuretic effect, which, in turn, increases sodium retention. NSAIDs also inhibit the vasodilating effects of prostaglandins and the production of vasoconstricting factors—namely, endothelin-1. These effects can contribute to the induction of hypertension in a normotensive or controlled hypertensive patient.

Endogenous hormonal causes include the following:

- Primary hyperaldosteronism - Cushing syndrome
- Pheochromocytoma - Congenital adrenal hyperplasia

Neurogenic causes include the following:

- Brain tumor - Bulbar poliomyelitis - Intracranial hypertension

Drugs and toxins that cause hypertension include the following:

- Alcohol - Cocaine - Cyclosporine, tacrolimus - NSAIDs
- Erythropoietin - Adrenergic medications - Decongestants (ephedrine)
- Herbal remedies containing licorice (including licorice root) or ephedrine (and ephedra)
- Nicotine

Other causes include the following:

- Hyperthyroidism and hypothyroidism - Hypercalcemia - Hyperparathyroidism
- Acromegaly - Obstructive sleep apnea - Pregnancy-induced HPN

Causes of hypertensive emergencies

The most common hypertensive emergency is a rapid unexplained rise in BP in a patient with chronic essential hypertension. Most patients who develop hypertensive emergencies have a history of inadequate hypertensive treatment or an abrupt discontinuation of their medications.

Patient Education

Hypertension is a lifelong disorder. For optimal control, a long-term commitment to lifestyle modifications and pharmacologic therapy is required.

Various strategies to decrease cardiovascular disease risk include the following:

- Prevention and treatment of obesity: an increase in body mass index (BMI) and waist circumference is associated with an increased risk of developing conditions with high cardiovascular risk, such as hypertension, diabetes mellitus, impaired fasting glucose, and left ventricular hypertrophy [LVH]
- Appropriate amounts of aerobic physical activity
- Diets low in salt, total fat, and cholesterol
- Adequate dietary intake of potassium, calcium, and magnesium
- Limited alcohol consumption
- Avoidance of cigarette smoking
- Avoidance of the use of illicit drugs, such as cocaine

History

Following the documentation of hypertension, which is confirmed after an elevated blood pressure (BP) on at least 3 separate occasions (based on the average of 2 or more readings taken at each of ≥2 follow-up visits after initial screening), a detailed history should extract the following information:

- Extent of end-organ damage (eg, heart, brain, kidneys, eyes)
- Assessment of patients' cardiovascular risk status
- Exclusion of secondary causes of hypertension

Patients may have undiagnosed hypertension for years without having had their BP checked. Therefore, a careful history of end-organ damage should be obtained. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) identifies the following as targets of end-organ damage:

- Heart: left ventricular hypertrophy, angina/previous myocardial infarction, previous coronary revascularization, and heart failure
- Brain: stroke or transient ischemic attack, dementia
- Chronic kidney disease
- Peripheral arterial disease
- Retinopathy

The JNC 7 identifies the following as major cardiovascular risk factor:

- Hypertension: component of metabolic syndrome
- Tobacco use, particularly cigarettes, including chewing tobacco
- Elevated LDL cholesterol (or total cholesterol ≥240 mg/dL) or low HDL cholesterol: component of metabolic syndrome
- Diabetes mellitus: component of metabolic syndrome
- Obesity (BMI ≥30 kg/m^2): component of metabolic syndrome
- Age greater than 55 years for men or greater than 65 years for women: increased risk begins at the respective ages; the Adult Treatment Panel III used earlier age cut points to suggest the need for earlier action
- Estimated glomerular filtration rate < 60 mL/min
- Microalbuminuria
- Family history of premature cardiovascular disease (men < 55 years; women < 65 years)

- Lack of exercise

Obtain a history of the patient's use of over-the-counter medications; herbal medicines such as herbal tea containing licorice عرق السوس

The historical and physical findings that suggest the possibility of secondary hypertension are a history of known renal disease, abdominal masses, anemia, and urochrome pigmentation.

- A history of sweating, labile hypertension, and palpitations suggests the diagnosis of pheochromocytoma.

- A history of cold or heat tolerance, sweating, lack of energy, and bradycardia or tachycardia may indicate hypothyroidism or hyperthyroidism.

- A history of obstructive sleep apnea may be noted. A history of weakness suggests hyperaldosteronism.

Kidney stones raise the possibility of hyperparathyroidism.

Physical Examination

An accurate measurement of blood pressure is the key to diagnosis. Several determinations should be made over a period of several weeks. At any given visit, an average of 3 blood pressure readings taken 2 minutes apart using a mercury manometer is preferable.On the first visit, blood pressure should be checked in both arms and in one leg to avoid missing the diagnosis of coarctation of aorta or subclavian artery stenosis.

The patient should rest quietly for at least 5 minutes before the measurement. Blood pressure should be measured in both the supine and sitting positions, auscultating with the bell of the stethoscope. As the improper cuff size may influence blood pressure measurement, a wider cuff is preferable, particularly if the patient's arm circumference exceeds 30 cm. Although somewhat controversial, the common practice is to document phase V (a disappearance of all sounds) of Korotkoff sounds as the diastolic pressure.

Ambulatory or home blood pressure monitoring provides a more accurate prediction of cardiovascular risk than do office blood pressure readings."Non-dipping" is the loss of the usual physiologic nocturnal drop in blood pressure and is associated with an increased cardiovascular risk.

A study by Wong and Mitchell indicated that independent of other risk factors, there is a link between the presence of certain signs of hypertensive retinopathy (eg, retinal hemorrhages, microaneurysms, cotton-wool spots) and an increased cardiovascular risk (eg, stroke, stroke mortality).Therefore, a funduscopic evaluation of the eyes should be performed to detect any evidence of early or late, chronic or acute hypertensive retinopathy, including arteriovenous nicking or changes in the vessel wall (eg, copper wiring, silver wiring, SOT , hard exudates, flame-shaped hemorrhages, papilledema). Indeed, ocular changes can be the initial finding in an asymptomatic patient necessitating a primary care referral; acute and chronic changes may manifest

in the eyes. Alternatively, a symptomatic patient may be referred to the ophthalmologist for visual changes due to hypertensive changes.

Palpation of all peripheral pulses should be performed. Absent, weak, or delayed femoral pulses suggests coarctation of the aorta or severe peripheral vascular disease. In addition, examine the neck for carotid bruits, distended veins, or enlarged thyroid gland. Listen for renal artery bruit over the upper abdomen; the presence of a bruit with both a systolic and diastolic component suggests renal artery stenosis.

A careful cardiac examination is performed to evaluate signs of LVH. These include displacement of apex, a sustained and enlarged apical impulse, and the presence of an S_4. Occasionally, a tambour S_2 is heard with aortic root dilatation.

Hypertensive Emergencies

The history and physical examination determine the nature, severity, and management of the hypertensive event. The history should focus on the presence of end-organ dysfunction. The physical examination should assess whether end-organ dysfunction is present (eg, neurologic, cardiovascular). BP should be measured in both the supine position and the standing position (assess volume depletion). BP should also be measured in both arms

(a significant difference may suggest aortic dissection).

The most common clinical presentations of hypertensive emergencies are cerebral infarction (24.5%), pulmonary edema (22.5%), hypertensive encephalopathy (16.3%), and congestive heart failure (12%). Other clinical presentations associated with hypertensive emergencies include intracranial hemorrhage, aortic dissection, and eclampsiaas well as acute myocardial infarction. Hypertension is also one of several conditions that have been increasingly recognized as having an association with posterior reversible encephalopathy syndrome (PRES), a condition characterized by headache, altered mental status, visual disturbances, and seizures.

Hypertensive Heart Disease

Uncontrolled and prolonged BP elevation can lead to a variety of changes in the myocardial structure, coronary vasculature, and conduction system of the heart. These changes in turn can lead to the development of left ventricular hypertrophy (LVH), coronary artery disease, various conduction system diseases, and systolic and diastolic dysfunction of the myocardium, which manifest clinically as angina ormyocardial infarction, cardiac arrhythmias (especially atrial fibrillation), andcongestive heart failure (CHF). Thus, hypertensive heart disease is a term applied generally to heart diseases—such as LVH, coronary artery disease, cardiac arrhythmias, and CHF—that are caused by direct orindirect effects of elevated BP.

Although these diseases generally develop in response to chronically elevated BP, marked and acute elevation of BP can also lead to accentuation of an underlying predisposition to any of the symptoms traditionally associated with chronic hypertension.

In a study by Tymchak et al, patients presenting with acute heart failure as a manifestation of hypertensive emergency were more likely to be black and have a history of heart failure; they were also more likely to have higher B-type natriuretic peptide (BNP) and creatinine levels and lower left ventricular ejection fraction. Note that BNP is inversely proportional to the degree of a patient's obesity.

Hypertension in Pediatric Patients

Systemic hypertension is less common in children than in adults, but the incidence of hypertension in children is approximately 1-5%. The presence of hypertension in younger children is usually indicative of an underlying disease process (secondary hypertension). In children, approximately 5-25% of cases of secondary hypertension are attributed to renovascular disease.

Hypertension in Pregnancy

Hypertension is the most common medical problem encountered during pregnancy, complicating 2-3% of pregnancies. Hypertensive disorders during pregnancy are classified into the 4 following categories, as recommended by the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy:

- Chronic hypertension
- Preeclampsia-eclampsia
- Preeclampsia superimposed on chronic hypertension
- pregnancy-induced hypertension (transient hypertension of pregnancy or chronic hypertension identified in the latter half of pregnancy.

Primary Aldosteronism

Mineralocorticoid excess secondary to primary hyperaldosteronism is infrequently observed and is characterized by excessive production of aldosterone. Renal sodium retention, kaliuresis, hypokalemia, and hypochloremic metabolic alkalosis are the common manifestations. It should be considered in patients who have an exaggerated hypokalemic response to a thiazide diuretic or who have hypokalemia unprovoked by a diuretic. These patients develop increased intravascular volume, resulting in hypertension. The BP increase may vary from mild hypertension to marked elevation in primary hyperaldosteronism. Patients may have underlying adenoma or hyperplasia of the adrenal gland and rarely have an extra-adrenal source for aldosterone.

In contrast, inappropriately high output of aldosterone for a given salt state of a patient (ie, not meeting criteria for a diagnosis of primary aldosteronism) is much more common, especially in patients with metabolic syndrome.

Approach Considerations

Many guidelines exist for the management of hypertension. Two of the most widely used recommendations are those from the American Diabetes Association (ADA) and the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7).

The ADA 2011 standards of medical care in diabetes also indicate that a majority of patients with diabetes mellitus have hypertension. In patients with type 1 diabetes,

nephropathy is often the cause of hypertension, whereas in type 2 diabetes, hypertension is one of a group of related cardiometabolic factors.Hypertension remains one of the most common causes of congestive heart failure (CHF). Antihypertensive therapy has been demonstrated to significantly reduce the risk of death from stroke and coronary artery disease.

JNC 7

Key messages of the JNC 7 are as follows:

- The goals of antihypertensive therapy is the reduction of cardiovascular and renal morbidity and mortality, with the focus on controlling the systolic BP, as most patients will achieve diastolic BP control when the systolic BP is achieved
- Prehypertension (systolic 120-139 mm Hg, diastolic 80-89 mm Hg) requires health-promoting lifestyle modifications to prevent the progressive rise in blood pressure and cardiovascular disease
- In uncomplicated hypertension, a thiazide diuretic, either alone or combined with drugs from other classes, should be used for the pharmacologic treatment of most cases
- In specific high-risk conditions, there are compelling indications for the use of other antihypertensive drug classes (eg, angiotensin-converting enzyme [ACE] inhibitors, angiotensin receptor blockers [ARBs], beta blockers, calcium channel blockers)
- Two or more antihypertensive medications will be required to achieve goal BP (< 140/90 mm Hg or < 130/80 mm Hg) for patients with diabetes and chronic kidney disease
- For patients whose BP is more than 20 mm Hg above the systolic BP goal or more than 10 mm Hg above the diastolic BP goal, initiation of therapy using 2 agents, one of which usually will be a thiazide diuretic, should be considered
- Regardless of therapy or care, hypertension will be controlled only if patients are motivated to stay on their treatment plan

Note that the JNC 8 is expected to be released in 2012.

Nonpharmacologic Therapy

Lifestyle modifications

JNC 7 and AHA-ASA lifestyle modification recommendations

Joint National Committee of Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7) recommendations to lower blood pressure :

- Weight loss helps to prevent hypertension
- (Dietary Approaches to Stop Hypertension) diet which is rich in fruits and vegetables and encourages the use of fat-free or low-fat milk and milk products
- Stop alcohol
- Reduce sodium intake
- Maintain adequate intake of dietary potassium يوجد بالموز
- Maintain adequate intake of dietary calcium and magnesium for general health
- Stop smoking and reduce intake of dietary saturated fat and cholesterol for overall cardiovascular health

- Exercise regularly.

The 2010 American Heart Association-American Stroke Association (AHA-ASA) guidelines for the primary prevention of stroke makes the following recommendations:

- **Hypertension**: the AHA-ASA guidelines recommend regular blood pressure screening, lifestyle modification, and drug therapy; lower risk of stroke and cardiovascular events are seen when systolic blood pressure levels are lower than 140 mm Hg and diastolic blood pressure levels are less than 90 mm Hg
- **Diet and nutrition**: a diet that is low in sodium and high in potassium is recommended to reduce BP; diets that promote the consumption of fruits, vegetables, and low-fat dairy products, help lower BP and may lower the risk of stroke
- **Physical inactivity**: increasing physical activity is associated with a reduction in the risk of stroke; the goal is to engage in 30 minutes or more of moderate intensity activity on a daily basis
- **Obesity and body fat distribution**: weight reduction in overweight and obese persons is recommended to reduce BP and the risk of stroke

Dietary changes

A number of studies have documented an association between sodium chloride intake and BP. The effect of sodium chloride is particularly important in individuals who are middle-aged to elderly with a family history of hypertension. A moderate reduction in sodium chloride intake can lead to a small reduction in blood pressure. The American Heart Association recommends that the average daily consumption of sodium chloride not exceed 6 g; this may lower BP by 2-8 mm Hg.

The DASH eating plan encompasses a diet rich in fruits, vegetables, and low-fat dairy products and may lower blood pressure by 8-14 mm Hg. The 2011 ADA standard of care supports the DASH diet, with the caution that high-quality studies of diet and exercise to lower blood pressure have not been performed on individuals with diabetes.

Weight loss and exercise

Weight reduction may lower blood pressure by 5-20 mm Hg per 10 kg of weight loss in a patient whose weight is more than 10% of ideal body weight.

Regular aerobic physical activity can facilitate weight loss, decrease BP, and reduce the overall risk of cardiovascular disease. Blood pressure may be lowered by 4-9 mm Hg with moderately intense physical activity. These activities include brisk walking for 30 minutes a day, 5 days per week. More intense workouts of 20-30 minutes, 3-4 times a week, may also lower BP and have additional health benefits.

The 2011 ADA diabetes standards support increasing physical activity. The recommendations emphasize that exercise is an important part of diabetes management in addition to reducing cardiovascular risk factors, contributing to weight loss, and improving overall well-being. Moreover, patients with diabetes and severe hypertension (SBP ≥140 mm Hg or DBP ≥90 mm Hg) at diagnosis or afterward should receive drug therapy along with lifestyle modifications.

Pharmacologic Therapy

If lifestyle modifications are insufficient to achieve the goal blood pressure (BP), there are several drug options for the treatment and management of hypertension.

The following are drug class recommendations for compelling indications based on various clinical trials:

- **Heart failure**: diuretic, beta-blocker, ACE inhibitor, ARB, aldosterone antagonist
- **Postmyocardial infarction**: beta-blocker, ACE inhibitor, aldosterone antagonist
- **High coronary disease risk**: diuretic, beta-blocker, ACE inhibitor, CCB
- **Diabetes**: diuretic, beta-blocker, ACE inhibitor, ARB, CCB
- **Chronic kidney disease**: ACE inhibitor, ARB
- **Recurrent stroke prevention**: diuretic, ACE inhibitor

Note that different stages of these diseases may alter their treatment management.

Multiple clinical trials suggest that most antihypertensive drugs provide the same degree of cardiovascular protection for the same level of BP control.

In addition, the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) study concluded that there were no differences in primary coronary heart disease outcome or mortality for the thiazide-like diuretic chlorthalidone, the ACE inhibitor lisinopril, and the CCB amlodipine. In a systematic review and meta-analysis, investigators also determined that in patients with essential hypertension without preexisting renal disease, no significant difference was found between Ras inhibitors and other antihypertensive agents in preventing renal dysfunction.

Management of Diabetes and Hypertension

Hypertension and diabetes are both risk factors for cardiovascular disease, stroke, progression of renal disease, and diabetic retinopathy.

In general, patients with diabetes type 1 or type 2 and hypertension have shown **clinical improvement with diuretics, ACE inhibitors, beta-blockers, ARBs, and calcium antagonists**. Most studies, however, have shown superiority of ACE inhibitors or ARBs over calcium antagonists in diabetic patients.

Two or more antihypertensive drugs at maximal doses should be used to achieve optimal BP targets in patients with diabetes and hypertension. Either an ACE inhibitor or an ARB is usually required in patients with diabetes and hypertension.

Management of Hypertensive Emergencies

Hypertensive emergencies are characterized by severe elevations in BP (>180/120 mm Hg) associated with acute end-organ damage.

Initial treatment goals are to reduce the mean arterial BP by no more than 25% within minutes to 1 hour. I

f the patient is stable, reduce the BP to 160/100-110 mm Hg within the next 2-6 hours. Several parenteral and oral therapies can be used to treat hypertensive emergencies, such as nitroprusside sodium, hydralazine, nicardipine, fenoldopam, nitroglycerin, or enalaprilat. Other agents that may be used include labetalol, esmolol, and

phentolamine. Avoid using short-acting nifedipine in the initial treatment of this condition because of the risk of rapid, unpredictable hypotension and the possibility of precipitating ischemic events.

Once the patient's condition is stabilized, the patient's BP may be gradually reduced over the next 24-48 hours.

Exceptions to the above recommendation include the following:

- Patients with an ischemic stroke (currently, no clear evidence exists for immediate antihypertensive treatment)
- Patients with aortic dissection (their systolic BP should be lowered to < 100 mm Hg, if tolerated)
- Patients in whom BP is lowered to allow thrombolytic therapy (eg, stroke patients)

Management of Hypertension in Pregnancy

In patients who are pregnant, the goal of antihypertensive treatment is to minimize the risk of maternal cardiovascular or cerebrovascular events. Hypertensive disorders—categorized as chronic hypertension, preeclampsia, chronic hypertension with superimposed preeclampsia, gestational hypertension, and transient hypertension (see Table 3, below)— may contribute to maternal, fetal, or neonatal morbidity and mortality, particularly in the first trimester.

Selection of antihypertensive medication

Although reducing maternal risk is the goal of treating chronic hypertension in pregnancy, it is fetal safety that largely directs the choice of antihypertensive agent. Methyldopa is generally the preferred first-line agent because of its safety profile. Other drugs that may be considered include labetalol, beta-blockers, and diuretics. Data are limited regarding the use of clonidine and calcium antagonists in pregnant women with chronic hypertension; however, angiotensin-converting enzyme (ACE) inhibitors and angiotensin II receptor (ARB) antagonists should be avoided because of the risk of fetal toxicity and death.

Management of Hypertension in the Elderly

Despite low plasma renin activity (PRA), blood pressure responds well to ACE inhibitor and ARB therapy. Low doses of diuretics may also be effective. Thiazide-type diuretics may be particularly beneficial for patients aged 55 years or older with hypertension or CVD risk factors and for patients aged 60 years or older with isolated systolic hypertension. The SHEP trial found that chlorthalidone stepped-care therapy for 4.5 years was associated with a longer life expectancy at 22-year follow-up in patients with isolated systolic hypertension. The Syst-Eur trial used a study design and sample size similar to those of the SHEP trial, in which treatment with the CCB nitrendipine resulted in significant reduction in stroke and overall CVD events.

Calcium antagonists are quite useful because of their strong antihypertensive effects. Often, combining 2 drugs at a lower dose may be preferable to using a single drug at a high dose, because of the potential for adverse effects with the higher dose. Beta-blockers may not be as effective as other first-line agents in patients aged 60 years and older, especially for stroke prevention, and should probably be used when other

indications are present, such as heart failure, previous myocardial infarction, and angina.

Elderly patients should also be encouraged to lose weight if necessary, be more physically active, reduce their salt intake, and avoid excessive alcohol intake.

According to the ACCF/AHA consensus, lifestyle modifications may be all that is necessary to treat milder forms of hypertension in elderly patients.However, drug treatment for elderly patients with hypertension is generally recommended and should be started at the lowest dose possible, with gradual increases depending on response.

Management of Ocular Hypertension

Hypertension, especially stage 2 hypertension, can affect the retina, choroid, and optic nerve, as well as increase intraocular pressure (IOP). In hypertensive retinopathy, the most common finding is generalized or focal narrowing of the retinal arterioles; occlusion or leakage of the retinal vessels may occur with acute or advanced hypertension. Hypertensive choroidopathy most commonly manifests in young patients with acute elevated blood pressure (BP), such as that which occurs in eclampsia or pheochromocytoma.

Treatment of ocular hypertension varies. Depending on the severity of the ocular hypertension, management may include observation or initiation of antihypertensive therapy. In general, pharmacologic treatment is initiated in patients who have an increased risk of developing glaucoma.

Blood pressure control may result in regression of signs of hypertensive retinopathy, but spontaneous resolution may also be possible.Among the issues that still need to be clarified are the following:

- Whether antihypertensive agents with potential direct beneficial microvascular effects (eg, ACE inhibitors) would reduce the damage of retinopathy beyond the reduction caused by lowered blood pressure
- Whether the specific reduction of hypertensive retinopathy also leads to reduction in cardiovascular disease morbidity and mortality
- Whether established risk-reducing interventions in targeted persons with hypertensive retinopathy would lead to additional advantages, as compared to the use of strategies without regard to retinal findings

In the presence of hypertensive optic neuropathy, a rapid reduction of BP may pose a risk of worsening ischemic damage to the optic nerve. The optic nerve demonstrates autoregulation, so there is an adjustment in perfusion based on BP. A precipitous reduction in BP will reduce perfusion to the optic nerve and central nervous system as a result of their autoregulatory changes, resulting in infarction of the optic nerve head and, potentially, acute ischemic neurologic lesions of the CNS.

Management of Renovascular Hypertension

The goals of therapy for renovascular hypertension (RVHT) are maintenance of normal blood pressure (BP) and prevention of end-stage renal disease (ESRD

It is important to note that the presence of renal artery stenosis or fibromuscular dysplasias are not always associated with renovascular hypertension.

ACE inhibitors are effective in patients with unilateral renal artery stenosis; however, ACE inhibitors need to be avoided in patients with bilateral renal artery stenosis or stenosis of a solitary kidney. A diuretic can be combined with an ACE inhibitor. Because of their glomerular vasodilatory effect, calcium antagonists are effective in renal artery stenosis and do not compromise renal function.

For most patients with RVHT, with the exception of persons with fibromuscular dysplasia, it is unclear whether revascularization will be beneficial. Fibromuscular dysplasia responds well to angioplasty. The causes of renovascular hypertension include atherosclerosis, fibromuscular dysplasia, coarctation of the aorta, embolic renal artery occlusion, aneurysm of the renal artery, and diffuse arteritis. Additionally, causes of diffuse bilateral renal ischemia (eg, accelerated hypertension, vasculitis, hepatitis B, and IV drug abuse) may also lead to hypertension.

Causes of resistant hypertension

Causes of resistant hypertension include improper BP measurement, volume overload, drug-induced or other causes, and associated conditions such as obesity or excessive alcohol intake.

Improper BP measurement

Improper BP measurement may result in falsely high readings, such as when the wrong-sized cuff is used, when patients have heavily calcified or arteriosclerotic brachial arteries, or in cases of white-coat hypertension (observed in 20-30% of patients).

Inadequate treatment and patient noncompliance

Inadequate treatment is common in cases of resistant hypertension ; in several published series, this has been described as the most common cause of resistant hypertension. Patients may not be on an effective drug or drug dose, or concomitant volume expansion may occur as a side effect of the drug.

Noncompliance with medical therapy or dietary modifications (eg, salt restriction) may play a role in causing resistant hypertension. Address noncompliance with extensive patient education, simplification of the drug regimen, use of fixed-dose combinations, and use of drugs with the fewest adverse effects.

Limited data suggest better compliance with ACE inhibitors and ARBs than with some of the other antihypertensive medications.

Extracellular volume expansion

Extracellular volume expansion may contribute to the inability to lower systemic BP. The volume expansion may occur because of renal insufficiency or because of sodium retention due to treatment with vasodilators, a high-salt diet, or insufficient dosing of a diuretic. This condition can be treated with more aggressive diuretic therapy until clinical signs of extracellular volume depletion (eg, orthostatic hypotension) develop.

The JNC 7 recommends a thiazide-type diuretic for the majority of hypertensive patients but notes that patients with a decreased GFR or who are in heart failure often require therapy with a loop diuretic.

Vasoactive substances

Resistant hypertension may be encountered in patients who are ingesting vasoactive substances despite taking antihypertensive drugs regularly. Salt and alcohol are common examples; others include cocaine, amphetamines, anabolic steroids, oral contraceptives, cyclosporine, antidepressants, and nonsteroidal anti-inflammatory drugs.

Excluding secondary causes

Whenever confronted with resistant hypertension, try to exclude any secondary causes of hypertension. A reevaluation of the patient's history, physical examination, and laboratory results may provide clues to secondary hypertension (eg, renal artery stenosis, primary hyperaldosteronism, obstructive sleep apnea). Primary hyperaldosteronism is estimated to have a prevalence of 20% in this population.

Management of Pseudohypertension

Pseudohypertension in an overestimation of intra-arterial pressure by cuff blood pressure (BP) measurement. This may be observed in elderly individuals who have thickened, calcified arteries, as the BP has relatively more difficulty compressing such arteries; much higher cuff pressure may be required to occlude a thickened brachial artery. The diastolic BP may also be overestimated.

Consider pseudohypertension in situations in which no organ damage occurs despite marked hypertension, when patients develop hypotensive symptoms on medications, and when calcification of the brachial artery is observed on radiologic examination. Direct measurement of intra-arterial pressure may be required in this setting.

Management of Primary Hyperaldosteronism

Hypokalemia (an unprovoked or an exaggerated hypokalemic response to a thiazide) and metabolic alkalosis are important clues to the presence of primary hyperaldosteronism. However, these are relatively late manifestations; in a large subset of patients, the serum potassium concentration and bicarbonate are within the reference range, and additional screening testing is needed in patients with high index of suspicion for primary hyperaldosteronism.

Measurement of the ratio of plasma aldosterone to renin activity ratio is the best initial screening test for primary hyperaldosteronism. A ratio of over 20-30 suggests that primary hyperaldosteronism may be present. Some labs require a minimum plasma aldosterone level of 12 ng/dL.

The diagnosis of primary hyperaldosteronism can be confirmed by the determination of the aldosterone excretion rate in a 24-hour urine following IV or oral salt loading (ie, urinary aldosterone excretion rate greater than 12-14 µg/24 hours, with urine sodium at

least 200 mEq/24 hours). Saline suppression testing can also be used to confirm the diagnosis.

The appropriate therapy depends on the cause of excessive aldosterone production. A CT scan with dynamic protocol may help localize an adrenal mass, indicating adrenal adenoma, which may be a nonsecreting incidentaloma or a hypersecreting adenoma. If the results of the CT scan are inconclusive, adrenal venous sampling for aldosterone and cortisol levels should be performed.

Medical therapy is indicated in patients with adrenal hyperplasia, patients with adenoma who are poor surgical risks, and patients with bilateral adenomas. These patients are best treated with sustained salt and water depletion. Hydrochlorothiazide or furosemide in combination with either spironolactone or amiloride corrects hypokalemia and normalizes the blood pressure. Some patients may require the addition of a vasodilator or a beta-blocker for better control of hypertension.

Adrenal adenomas may be resected via a laparoscopic procedure. Surgical resection often leads to the control of blood pressure and the reversal of biochemical abnormalities. These patients may develop hypoaldosteronism during the postoperative follow-up period and require supplementation with fludrocortisone.

Prevention

Prevention of hypertension may be achieved by the following interventions:

- Weight control
- Increased physical activity
- Moderated sodium and alcohol intake
- Increased potassium intake
- A diet rich in fruits and vegetables and low-fat meat, fish, and dairy products

Medication Summary

Many therapeutic agents can be used for the pharmacologic management of hypertension. The general recommendation established by JNC-7 is to initiate a thiazide-type diuretic initially for stage 1 hypertensives without compelling indications for other therapies.

Drugs such as angiotensin converting enzyme (ACE) inhibitors, calcium channel blockers (CCBs), angiotensin receptor blockers (ARBs), beta-blockers, and diuretics are all considered acceptable alternative therapies in patients with hypertension.

The available antihypertensive agents are generally equally effective in lowering blood pressure however; there may be interpatient variability that can affect the way a patient will respond to one treatment over another.

Diuretics, Thiazide

Class Summary

Thiazide diuretics are used as monotherapy, or they can be administered adjunctively with other antihypertensive agents. Thiazide diuretics inhibit reabsorption of sodium

and chloride mostly in the distal tubules. Long-term use of these drugs may result in hyponatremia.

They also increase potassium and bicarbonate excretion and decrease calcium excretion and uric acid retention. Thiazides do not affect normal blood pressure.

Keep in mind that all available loop and thiazide diuretic agents, except ethacrynic acid, possess a sulfonamide group, which has important clinical relevance to those individuals with allergies to sulfonamide agents.

Hydrochlorothiazide

Hydrochlorothiazide is approved for the management of hypertension, alone or in combination with other antihypertensive agents. Unlike potassium-sparing combination diuretic products, hydrochlorothiazide may be used in patients who cannot risk the development of hyperkalemia, including patients taking ACE inhibitors.

Hydrochlorothiazide is available as oral tablets or capsules in doses ranging from 12.5-50 mg. The usual dose is 12.5 mg given alone or in combination with other antihypertensives, with a maximum dose of 50 mg daily. Doses greater than 50 mg are associated with hypokalemia.

Chlorthalidone

Chlorthalidone is indicated for the management of hypertension either alone or in combination with other antihypertensives. The initial dosage is 25 mg as a single daily dose. Dosage can be titrated to 50 mg if the clinical response is not adequate. If additional control is required, increase the dosage to 100 mg once daily, or a second antihypertensive drug may be added. Doses greater than 100 mg daily usually do not increase effectiveness. Increases in serum uric acid and hypokalemia are dose-related over the 25-100 mg/day range.

Metolazone

Metolazone is approved for the treatment of hypertension either alone (uncommon) or in combination with other antihypertensives. The initial dosage for hypertension is 2.5 to 5 mg given once daily. Metolazone does not decrease glomerular filtration rate or the renal plasma flow and may be a more effective option for patients with impaired renal function.

Indapamide

Indapamide is chemically not a thiazide, although its structure and function are very similar. The drug enhances the excretion of sodium, chloride, and water by inhibiting the transport of sodium ions across the renal tubule.

The hypovolemic action of indapamide is believed to be responsible for the drug's beneficial cardiovascular effects. The half-life of indapamide is approximately 14 hours, so the drug can be taken just once daily. Adverse effects tend to be somewhat milder than with thiazides.

Diuretic, Potassium-Sparing

Class Summary

The potassium-sparing diuretics interfere with sodium reabsorption at the distal tubules (primarily in the collecting duct region of the nephron), decreasing potassium secretion. Potassium-sparing diuretics have a weak diuretic and antihypertensive effect when used alone.

Triamterene

Triamterene is used alone or with other medications (often a kaliuretic diuretic such as hydrochlorothiazide) to treat edema and high blood pressure. Because triamterene increases potassium levels, caution is required when combining triamterene with ACE inhibitors, angiotensin receptor blockers, aliskiren, and other drugs that increase potassium levels. Potassium level should be monitored at start of treatment, dose change, and during illness that affects renal function. The recommended dose is 100 mg twice daily (maximum dose is 300 mg/d).

Amiloride

Amiloride is a potassium-conserving (antikaliuretic) drug that, compared with thiazide diuretics, possesses weak natriuretic, diuretic, and antihypertensive activity. It is approved as adjunctive treatment with thiazide diuretics or other kaliuretic-diuretic agents for hypertension or congestive heart failure. It is unrelated chemically to other known antikaliuretic or diuretic agents. Amiloride has little additive diuretic or antihypertensive effect when added to a thiazide diuretic. Amiloride can be given at a dose of 5-10 mg daily in 1-2 divided doses for hypertension. Amiloride has a black box warning for hyperkalemia, which, if not corrected, is potentially fatal. This incidence is greater in patients with renal impairment or diabetes mellitus and in the elderly.

Diuretics, Loop

Class Summary

Loop diuretics act on the ascending limb of the loop of Henle, inhibiting the reabsorption of sodium and chloride. The loop diuretics are highly protein-bound and therefore enter the urine primarily by tubular secretion in the proximal tubule, rather than by glomerular filtration.

Loop diuretics are commonly used to control volume retention. Generally, thiazide diuretics are recommended for most patients with a diagnosis of hypertension; however, loop diuretics are more commonly prescribed for patients with decreased glomerular filtration rate or heart failure. Loop diuretics do not reduce blood pressure as effectively as thiazide diuretics when they are used as monotherapy, especially if they are dosed once daily.

Keep in mind that all available loop and thiazide diuretic agents, except ethacrynic acid, possess a sulfonamide group, which has important clinical relevance to those individuals with allergies to sulfonamide agents.

Furosemide (Lasix)

Furosemide is approved for the treatment of hypertension alone (uncommon) or in combination with other antihypertensive agents. Hypertensive patients who cannot be adequately controlled with thiazides will probably also not be adequately controlled with furosemide alone. The initial dosing recommendations for hypertension are usually 80 mg (divided into 40 mg twice a day). If clinical response is not sufficient, additional antihypertensives may be added. Patients should be monitored carefully because furosemide is a potent diuretic. If given in excessive amounts, it can cause profound diuresis with water and electrolyte depletion. Furosemide is available as an oral tablet and injection solution.

Torsemide

Torsemide can be used as monotherapy or in combination with other antihypertensive agents. The initial dose is 5 mg once daily. The dose can be titrated to 10 mg once daily. If adequate response is not seen, an additional antihypertensive agent may be needed. Torsemide is available as an oral tablet and injection solution.

Bumetanide

Bumetanide is FDA approved for the treatment of edema. It is also used off-label for the treatment of hypertension. The usual dosage range for bumetanide for hypertension is 0.5-2 mg/day given once or twice a day.

ACE Inhibitors

Class Summary

Angiotensin converting enzyme (ACE) inhibitors are the treatment of choice in patients with hypertension, chronic kidney disease, and proteinuria. ACE inhibitors reduce morbidity and mortality rates in patients with heart failure, patients with recent myocardial infarctions, and patients with proteinuric renal disease. ACE inhibitors appear to act primarily through suppression of the renin-angiotensin-aldosterone system. ACE inhibitors prevent the conversion of angiotensin I to angiotensin II and block the major pathway of bradykinin degradation by inhibiting ACE. Accumulation of bradykinin has been proposed as an etiologic mechanism for the side effects of cough and angioedema. ACE inhibitors can cause injury or even death to a developing fetus. In pregnant patients, ACE inhibitors should be discontinued as soon as possible.

It is important to note that the blood-pressure-lowering effects of ACE inhibitors and thiazides are approximately additive, and there is also the potential for hyperkalemia when ACE inhibitors are coadministered with potassium supplements or potassium-sparing diuretics. In addition, a study by Harel et al found an increased risk for hyperkalemia when aliskiren, a direct renin inhibitor, and ACE inhibitors or angiotensin receptor blockers were used together. Careful monitoring of serum potassium levels is warranted when these agents are used in combination. Furthermore, in patients with hypertension plus type 2 diabetes and renal impairment who are at high risk of cardiovascular and renal events, there is an increased risk of nonfatal stroke, renal complications, hypokalemia, and hypotension when aliskiren is added to ACE inhibitor or ARB therapy.

Fosinopril

Fosinopril may be used alone or in combination with other antihypertensive agents. Initial dose is 5 mg daily up to a maximum of 40 mg daily. May be divided into twice daily dosing. Unlike most ACE inhibitors that are primarily excreted by the kidneys, fosinopril is eliminated by both renal and hepatic pathways, making it a safer choice in patients with renal failure and heart failure patients with impaired kidney function.

Captopril

Captopril is indicated for the treatment of hypertension. It can be used alone or in combination with other antihypertensive drugs, such as diuretics or beta-adrenergic-blocking agents. The initial dose is 25 mg given 2 to 3 times daily. If reduction of blood pressure is not achieved after 1 or 2 weeks, the dose can be titrated to 50 mg 2 or 3 times daily. If further blood reduction is required after addition of a diuretic, the dose of captopril may be increased to 100 mg 2 or 3 times daily and then, if necessary, to 150 mg 2 or 3 times daily (while continuing the diuretic).

Ramipril

Ramipril is indicated for the treatment of hypertension alone or in combination with thiazide diuretics. The initial dosing recommendation for ramipril is 2.5 mg daily for patients who are not receiving a diuretic. Doses can range from 2.5-20 mg/day given once or twice a day.

Enalapril

Enalapril is effective alone or in combination with other antihypertensive agents, especially thiazide-type diuretics. The initial dose of enalapril is 5 mg daily. Dosage can range from 10-40 mg/day administered as a single dose or in 2 divided doses.

Lisinopril

Lisinopril may be used as monotherapy or concomitantly with other classes of antihypertensive agents. The initial dose of lisinopril is 10 mg daily. The dosage can range from 20-40 mg/day as a single daily dose. Doses up to 80 mg/day have been used; however, they do not show a greater effect.

Quinapril

Quinapril may be used alone or in combination with thiazide diuretics. The initial dose is 10 to 20 mg daily for patients not on diuretics. If blood pressure is not controlled with quinapril monotherapy, adding a diuretic should be considered.

ARBs

Class Summary

Generally, ACE inhibitors should remain the initial treatment of choice for hypertension. Angiotensin II receptor antagonists or angiotensin receptor blockers (ARBs) are used for patients who are unable to tolerate ACE inhibitors. ARBs competitively block binding of angiotensin-II to angiotensin type I (AT_1) receptors, thereby reducing effects of angiotensin II–induced vasoconstriction, sodium retention, and aldosterone release;

the breakdown of bradykinin should not be inhibited. If monotherapy with an ARB is not sufficient, adding a diuretic should be considered.

Losartan

Losartan may be used alone or in combination with other antihypertensive agents, including diuretics. The initial dose is 50 mg daily; however, in patients on diuretic therapy, the initial dose is 25 mg daily. A low-dose diuretic (eg, hydrochlorothiazide) may be added if blood pressure is not controlled. Losartan can be titrated up to 100 mg daily.

Valsartan

Valsartan is approved for the treatment of hypertension in adults and in children 6-16 years of age. It may be used alone or in combination with other antihypertensive agents. The initial dose is 80 or 160 mg once daily when used as monotherapy in patients who are not volume depleted. The valsartan dose may be increased (maximum 320 mg/day), or a diuretic may be added if additional blood pressure reduction is required. The addition of a diuretic has a greater effect than dose increases above 80 mg.

Beta-Blockers, Beta-1 Selective

Class Summary

Beta-blockers are generally not recommended as first-line agents for the treatment of hypertension; however, they are suitable alternatives when a compelling cardiac indication (eg, heart failure, myocardial infarction, diabetes) is present. Selective beta-blockers specifically block beta-1 receptors alone, although they can be nonselective at higher doses.

Caution should be used in administering these agents in the setting of asthma or severe chronic obstructive pulmonary disease (COPD), regardless of beta-selectivity profile. In addition, exacerbations of angina and, in some cases, myocardial infarction have been reported following abrupt discontinuance of beta-blocker therapy. The doses should be gradually reduced over at least a few weeks.

Atenolol

Atenolol is approved for the management of hypertension used alone or concomitantly with other antihypertensive agents, particularly with a thiazide-type diuretic. The initial dose is 50 mg daily, alone or added to diuretic therapy. If adequate clinical effect is not seen, the dose can be titrated to 100 mg daily. Other studies suggest that atenolol lacks specific potential for stroke reduction.

Metoprolol

Metoprolol is approved for the management of hypertension alone or concomitantly with other antihypertensive agents. The initial dose for metoprolol immediate release is 100 mg daily in single or divided doses, with or without a diuretic (maximum 450 mg/day). Metoprolol extended-release formulation can be started at a dose of 25-100 mg daily in a single dose, with or without a diuretic (maximum 400 mg/day).

Propranolol (Inderal)

Propranolol is approved for the management of hypertension alone or concomitantly with other antihypertensive agents. The initial dose is 40 mg given twice daily, alone or added to diuretic therapy. Dose can be titrated based on a patient's clinical response. The maintenance dose can range from 120-240 mg/day (maximum 640 mg/day). Exacerbations of angina and, in some cases, myocardial infarction, following abrupt discontinuance of propranolol therapy have been reported. The propranolol dose should be gradually reduced over at least a few weeks.

Bisoprolol

Bisoprolol is approved for the management of hypertension alone or in combination with other antihypertensive agents. This agent is a more specific beta-1 blocker than other beta-blockers. The initial dose is 5 mg once daily (reduce to 2.5 mg for patients with bronchospastic disease). The dosage can be titrated to 10 mg/day and then to 20 mg/day if necessary.

Timolol

Timolol is indicated for the treatment of hypertension. It is used alone or in combination with other antihypertensive agents, especially thiazide-type diuretics. The initial dose is 10 mg given twice daily. The total daily dose can be titrated to a maximum of 30 mg administered in divided doses. Avoid abrupt cessation of therapy, because of the risk of exacerbation of ischemic heart disease.

Beta-Blockers, Alpha Activity

Class Summary

Beta-blockers, such as labetalol and carvedilol, have peripheral vasodilatory effects that act via antagonism of the alpha-1 receptor in addition to beta-receptors.

Labetalol

Labetalol is indicated for the management of hypertension. Labetalol tablets may be used alone or in combination with other antihypertensive agents, especially thiazide and loop diuretics. The initial dose is 100 mg given twice daily. The dose may be titrated after 2-3 days in increments of 100 mg twice a day every 2-3 days (maximum 2400 mg/day).

Labetalol's actions at alpha-1 and beta-receptors lead to vasodilation and decreased total peripheral resistance, which results in decreased blood pressure without a substantial decrease in resting heart rate, cardiac output, or stroke volume.

Carvedilol

Carvedilol is approved for the management of essential hypertension. It can be used as monotherapy or in combination with other antihypertensive agents, especially thiazide-type diuretics. The initial dose is 6.25 mg given twice daily. The dose can be titrated at intervals of 7-14 days to 12.5 mg twice daily, then to 25 mg twice daily as needed (maximum 50 mg/day).

Similar to labetalol, carvedilol antagonizes both alpha-1 and beta-receptors. Carvedilol lowers standing blood pressure more than supine blood pressure; orthostatic hypotension may occur.

Beta-Blockers, Intrinsic Sympathomimetic

Class Summary

Agents such as acebutolol and pindolol possess intrinsic sympathomimetic activity (ISA). These agents can be used alone or in combination with other antihypertensive agents, particularly with a thiazide-type diuretic.

Vasodilators

Vasodilators relax blood vessels to improve blood flow, thus decreasing blood pressure.

Hydralazine

Oral hydralazine is indicated for essential hypertension, alone or as an adjunct. Initial dose is 10 mg given 4 times daily for the first 2 to 4 days, then 25 mg 4 times a day for 1 week. Hydralazine IV or IM is indicated for severe essential hypertension when the drug cannot be given orally or when there is an urgent need to lower BP.

Hydralazine may lower blood pressure by exerting a peripheral, vasodilating effect through a direct relaxation of vascular smooth muscle. Caution should be used when hydralazine is administered in patients with concomitant coronary artery disease.

Minoxidil

Minoxidil is indicated in severe hypertension that is symptomatic or associated with end-organ damage and is not manageable with maximum therapeutic doses of a diuretic plus 2 other antihypertensives. The initial dose is 5 mg/day as a single dose and can be titrated to 10, 20, and then 40 mg in single or divided doses as needed (maximum 100 mg/day). Minoxidil reduces elevated systolic and diastolic blood pressure by decreasing peripheral vascular resistance. The blood pressure response to minoxidil is dose-related and proportional to the extent of hypertension. Concomitant therapy with an antiadrenergic agent and loop diuretic is generally required.

Calcium Channel Blockers

Calcium channel blockers (CCBs) can be divided into dihydropyridines and nondihydropyridines. Dihydropyridines bind to L-type calcium channels in the vascular smooth muscle, which results in vasodilatation and a decrease in blood pressure. They are effective as monotherapy in black patients and elderly patients. Some examples of dihydropyridines include amlodipine, nifedipine, clevidipine, and felodipine. Non-dihydropyridines such as verapamil and diltiazem bind to L-type calcium channels in the sinoatrial and atrioventricular node, as well as exerting effects in the myocardium and vasculature. These agents may constitute a more effective class of medication for black patients.

Nifedipine

Nifedipine extended-release is indicated for the treatment of hypertension alone or in combination with other antihypertensive agents. The usual dose for nifedipine is 30-60 mg once daily (maximum 90 mg/day); when used for hypertension, nifedipine can be administered to a maximum of 120 mg/day.

Amlodipine

Amlodipine is a dihydropyridine CCB that has antianginal and antihypertensive effects. Amlodipine is a peripheral arterial vasodilator that acts directly on vascular smooth muscle to cause a reduction in peripheral vascular resistance and reduction in blood pressure.

Diltiazem

Diltiazem is a nondihydropyridine CCB that produces its antihypertensive effect primarily by relaxation of vascular smooth muscle and the resultant decrease in peripheral vascular resistance. The magnitude of blood pressure reduction is related to the degree of hypertension.

Verapamil

Verapamil is a nondihydropyridine that produces its antihypertensive effect by a combination of vascular and cardiac effects. It acts as a vasodilator with selectivity for the arterial portion of the peripheral vasculature. As a result, the systemic vascular resistance is reduced, usually without orthostatic hypotension or reflex tachycardia.

Aldosterone Antagonists, Selective

Aldosterone antagonists compete with aldosterone receptor sites, reducing blood pressure and sodium reabsorption.

Spironolactone

Spironolactone is usually used in combination with other drugs for patients who cannot be treated adequately with other agents or for whom other agents are considered inappropriate. The initial dose ranges from 50-100 mg daily in single or divided doses. Spironolactone can cause hyperkalemia; therefore, potassium supplementation should not be given concurrently. Other adverse effects include gynecomastia and impotence, which often mitigates the use of spironolactone in younger men.

Alpha$_2$-Agonists, Central-Acting

Centrally acting alpha2-agonists stimulate presynaptic alpha2-adrenergic receptors in the brain stem, which reduces sympathetic nervous activity.

Methyldopa

Methyldopa stimulates central alpha-adrenergic receptors by a false transmitter, exerting a direct effect on peripheral sympathetic nerves. Decreases in blood pressure are greatest when the patient is standing but are also significant when the patient is

supine. Postural hypotension has been reported in patients receiving methyldopa. Methyldopa is not associated with a rebound effect, as with clonidine.

Clonidine

Clonidine stimulates alpha2-adrenoreceptors in the brain stem, activating an inhibitory neuron, which in turn results in reduced sympathetic outflow. These effects result in a decrease in peripheral resistance, renal vascular resistance, blood pressure, and heart rate. Clonidine can be used alone or in combination with other antihypertensives. Clonidine is associated with a rebound effect, especially at higher doses or with more severe hypertension.

Renin Inhibitors/Combos

Renin inhibitors act within the renin-angiotensin system (RAS), a hormone system important in the regulation of blood pressure, electrolyte homeostasis, and vascular growth. Renin inhibitors have an additive effect when used with diuretics. Avoid the use of these agents in pregnancy.

Aliskiren

Aliskiren decreases plasma renin activity and inhibits conversion of angiotensinogen to angiotensin I (as a result, also decreasing angiotensin II) and thereby disrupts the renin-angiotensin-aldosterone system feedback loop. It is indicated for hypertension as monotherapy or in combination with other antihypertensive drugs. This agent remains under investigation.

Alpha-Blockers, Antihypertensives

Alpha-blockers are generally not recommended as initial monotherapy. They selectively block postsynaptic $alpha_1$ -adrenergic receptors. They dilate arterioles and veins, thus lowering blood pressure. These drugs can be combined with any of the other antihypertensives in other drug classes. Common side effects seen in this drug class include dizziness, headache, and drowsiness, in addition to orthostatic and first-dose hypotension.

Prazosin (Minipress)

Prazosin is a competitive antagonist at postsynaptic alpha1-receptors. Prazosin causes peripheral vasodilation by selective, competitive inhibition of vascular postsynaptic alpha1-adrenergic receptors, thus reducing peripheral vascular resistance and blood pressure.

Terazosin

Terazosin causes peripheral vasodilation by selective, competitive inhibition of vascular postsynaptic alpha1-adrenergic receptors, thereby reducing peripheral vascular resistance and blood pressure. Terazosin reduces blood pressure in both the supine and the standing positions, with more dramatic effects on diastolic blood pressure.

Doxazosin

Doxazosin is a selective alpha1-adrenergic antagonist. It inhibits postsynaptic alpha-adrenergic receptors, resulting in vasodilation of veins and arterioles and a decrease in total peripheral resistance and blood pressure. The antihypertensive effect of doxazosin mesylate results from a decrease in systemic vascular resistance.

Antihypertensives, Other

Reserpine is a peripherally acting adrenergic agent. It is indicated for mild hypertension and can be used as adjunctive therapy with other antihypertensive agents in more severe forms of hypertension.

Reserpine

Reserpine reduces blood pressure by depleting sympathetic biogenic amines.

The result of reserpine's effects on biogenic amines is sympathetic dysfunction, with a subsequent decrease in peripheral vascular resistance and a lowering of blood pressure often associated with bradycardia. This agent is also associated with depression.

Antihypertensive Combinations

Drug combinations using agents that act by different mechanisms have an additive effect. Most clinicians recommend initiating therapy with a single agent and advancing to the low-dose combination therapy. Some patients will require multiple medications to achieve their blood pressure targets and will benefit from drug combinations. Drug combination therapy may also help to improve patient compliance.

Metoprolol/hydrochlorothiazide

Metoprolol/hydrochlorothiazide is a combination of metoprolol, a beta-blocker, and hydrochlorothiazide, a thiazide diuretic. Metoprolol is a beta1-selective blocker at low doses; at higher doses, it also inhibits beta2-adrenoreceptors. Hydrochlorothiazide inhibits sodium reabsorption in distal renal tubules, resulting in increased excretion of water, sodium, potassium, and hydrogen ions.

Valsartan/hydrochlorothiazide

Valsartan/hydrochlorothiazide is a combination of valsartan, an angiotensin receptor blocker, and hydrochlorothiazide, a diuretic. Valsartan is a prodrug that produces direct antagonism of angiotensin II receptors. It displaces angiotensin II from AT1 receptor and may lower blood pressure by antagonizing AT1-induced vasoconstriction, aldosterone release, catecholamine release, arginine vasopressin release, water intake, and hypertrophic responses. Hydrochlorothiazide inhibits sodium and chloride reabsorption in distal renal tubules, resulting in increased excretion of water, sodium, potassium, and hydrogen ions.

Valsartan/amlodipine/hydrochlorothiazide

Valsartan/amlodipine/hydrochlorothiazide is a combination of amlodipine, a dihydropyridine calcium channel blocker, valsartan, an angiotensin receptor blocker,

and hydrochlorothiazide, a diuretic. Amlodipine exhibits antianginal and antihypertensive effects by inhibiting the influx of calcium in cardiac and smooth muscle cells of the coronary and peripheral vasculature, resulting in dilatation of coronary and peripheral arteries. Valsartan is a prodrug that produces direct antagonism of angiotensin II receptors. It displaces angiotensin II from the AT1 receptor and may lower blood pressure by antagonizing AT1-induced vasoconstriction, aldosterone release, catecholamine release, arginine vasopressin release, water intake, and hypertrophic responses. Hydrochlorothiazide inhibits sodium and chloride reabsorption in distal renal tubules, resulting in increased excretion of water, sodium, potassium, and hydrogen ions.

Enalapril/hydrochlorothiazide

Enalapril/hydrochlorothiazide is a combination of enalapril, an ACE inhibitor, and hydrochlorothiazide, a diuretic. Hydrochlorothiazide inhibits sodium reabsorption in distal renal tubules, resulting in increased excretion of water, sodium, potassium, and hydrogen ions. Enalapril prevents the conversion of angiotensin I to angiotensin II, a potent vasoconstrictor, resulting in increased levels of plasma renin and a reduction in aldosterone secretion. It helps control blood pressure and proteinuria.

Malignant hypertension

Malignant hypertension and accelerated hypertension are both hypertensive emergencies, with similar outcomes and therapies. Malignant hypertension may or may not be associated with clinical conditions present in hypertensive urgency. A patient with malignant hypertension always has retinal papilledema (as seen in the image below), as well as flame-shaped hemorrhages and exudates. Other clinical features of malignant hypertension may include encephalopathy, confusion, left ventricular failure, intravascular coagulation, and impaired renal function, with hematuria and weight loss.

The pathologic hallmark of malignant hypertension is fibrinoid necrosis of the arterioles, which occurs systemically, but specifically in the kidneys. These patients develop fatal complications if untreated, and more than 90% will not survive beyond 1-2 years..



Papilledema. Note the swelling of the optic disc, with blurred margins

Management of Hypertensive Emergencies

Approximately 3-45% of adult patients have at least one incident of increased BP during their stay in the emergency department (ED). The fundamental principle in determining the necessary ED care of the hypertensive patient is the presence or absence of end-organ dysfunction. Many patients present to the ED with elevated BPs; however, only a small proportion of patients will require emergency treatment. An important point to remember in the management of the patient with any degree of BP elevation is to "treat the patient and not the number."

The primary goal of the emergency physician is to determine which patients with acute hypertension are exhibiting symptoms of end-organ damage and require immediate intravenous (IV) parenteral therapy. In contrast, patients presenting with acutely elevated BP (systolic BP [SBP] >200 mm Hg or diastolic BP [DBP] >120 mm Hg) without symptoms that are sustained throughout the ED stay and whose BP stays significantly elevated to this level on discharge should have initiation of medical therapy and close follow-up in the outpatient setting.

Thus, optimal control of hypertensive situations balances the benefits of immediate decreases in BP against the risk of a significant decrease in target organ perfusion. The emergency physician must be capable of appropriately evaluating patients with an elevated BP, correctly classifying the hypertension, determining the aggressiveness and timing of therapeutic interventions, and making disposition decisions.

Acutely lowering BP in the ED for clinical situations other than those listed below is controversial and generally should be avoided.

Pharmacotherapy

An important point to remember in the management of the patient with any degree of BP elevation is to "treat the patient and not the number." In patients presenting with hypertensive emergencies, antihypertensive drug therapy has been shown to be effective in acutely decreasing blood pressure.

Sodium nitroprusside is a commonly used medication. It is a short-acting agent, and the BP response can be titrated from minute to minute. However, patients must have constant monitoring in an intensive care unit. The potential exists for thiocyanate and cyanide toxicity with prolonged use or if the patient has renal orhepatic failure.

Diazoxide may also be used in hypertensive crisis. Small boluses of 100 mg are administered every 5 minutes, as indicated. Diazoxide is not preferred with concomitant congestive heart failure or low cardiac output.

Labetalol, an alpha- and beta-blocking agent, has proven to be quite beneficial in the treatment of patients with hypertensive emergencies. Labetalol is particularly preferred in patients with acute dissection. Boluses of 10-20 mg may be administered, or the drug may be infused at 1 mg/min until the desired BP is obtained. Once an adequate BP level is obtained, oral hypertensive therapy should be initiated, and patients are gradually weaned from parenteral agents.

As the BP approaches its goal, increase the clevidipine dose by less than double, and lengthen the time between dose adjustments to every 5-10 minutes. An approximately 1-2 mg/h increase produces an additional 2-4 mm Hg decrease in SBP. Typically, the

therapeutic response is achieved with 4-6 mg/h, although severe hypertension may require higher doses. Most patients have received maximum doses of 16 mg/h or less; experience is limited with short-term dosing as high as 32 mg/h. Because of lipid load restrictions, do not exceed 1000 mL or an average of 21 mg/h within a 24-hour period; experience is limited with use beyond 72 hours.

Neurologic emergencies

Rapid BP reduction is indicated in neurologic emergencies, such as hypertensive encephalopathy, acute ischemic stroke, acute intracerebral hemorrhage, andsubarachnoid hemorrhage.

In hypertensive encephalopathy, the treatment guidelines are to reduce the MAP 25% over 8 hours. Labetalol, nicardipine, esmolol are the preferred medications; however, nitroprusside and hydralazine should be avoided.

For acute ischemic stroke, the preferred medications are labetalol and nicardipine. Withhold antihypertensive medications unless the SBP is >220 mm Hg or the DBP is >120 mm Hg, UNLESS the patient is receiving IV or intra-arterial (IA) fibrinolysis; then, the goal BP is an SBP of < 185 mm Hg and DBP < 110 mm Hg. After treatment with fibrinolysis, the SBP should be maintained < 180 mm Hg and the DBP at < 105 mm Hg for 24 hours.

For acute intracerebral hemorrhage, the preferred medications are labetalol, nicardipine, and esmolol; avoid nitroprusside and hydralazine. The treatment is based on clinical/radiographic evidence of increased intracranial pressure (ICP). If there are signs of increased ICP, maintain the MAP just below 130 mm Hg (or SBP < 180 mm Hg) for the first 24 hours after onset. In patients without increased ICP, maintain the MAP < 110 mm Hg (or SBP < 160 mm Hg) for the first 24 hours after symptom onset.

Recent evidence shows that in cases of acute intracerebral hemorrhage, early intensive BP control is well tolerated and can reduce hematoma growth in patients treated within 6 hours after the onset of intracerebral hemorrhage. The target systolic pressure for these studies was 140 mm Hg and routine IV medications were used. The target SBP was maintained over 7 days.

In subarachnoid hemorrhage, nicardipine, labetalol, and esmolol are also the preferred agents; again, nitroprusside and hydralazine should be avoided. Maintain the SBP < 160 mm Hg until the aneurysm is treated or cerebral vasospasm occurs. Although oral nimodipine is used to prevent delayed ischemic neurologic deficits, it is NOT indicated for treating acute hypertension.

Cardiovascular emergencies

Rapid BP reduction is also indicated in cardiovascular emergencies, such as aortic dissection, acute coronary syndrome, and acute heart failure.

In aortic dissection, the preferred medications are labetalol, nicardipine, nitroprusside (with beta-blocker), esmolol, and morphine sulfate. However, avoid beta-blockers if there is aortic valvular regurgitation or suspected cardiac tamponade. Maintain the SBP at < 110 mm Hg, unless signs of end-organ hypoperfusion are present. The

preferred treatment includes a combination of narcotic analgesics (morphine sulfate), beta blockers (labetalol, esmolol), and vasodilators (nicardipine, nitroprusside). Calcium channel blockers (verapamil, diltiazem) are an alternative to beta blockers.

For acute coronary syndrome, beta blockers and nitroglycerin are the preferred drugs. Treatment is indicated if the SBP is >160 mm Hg and/or the DBP is >100 mm Hg. Reduce the BP by 20-30% of baseline. Note that thrombolytics are contraindicated if the BP is >185/100 mm Hg.

In acute heart failure, the preferred medications are IV or sublingual nitroglycerin and IV enalaprilat. Treat with vasodilators (in addition to diuretics) for a SBP ≥140 mm Hg.

Cocaine toxicity/pheochromocytoma

Diazepam, phentolamine, and nitroglycerin/nitroprusside are the preferred drugs. However, avoid beta-adrenergic antagonists before administering phentolamine.

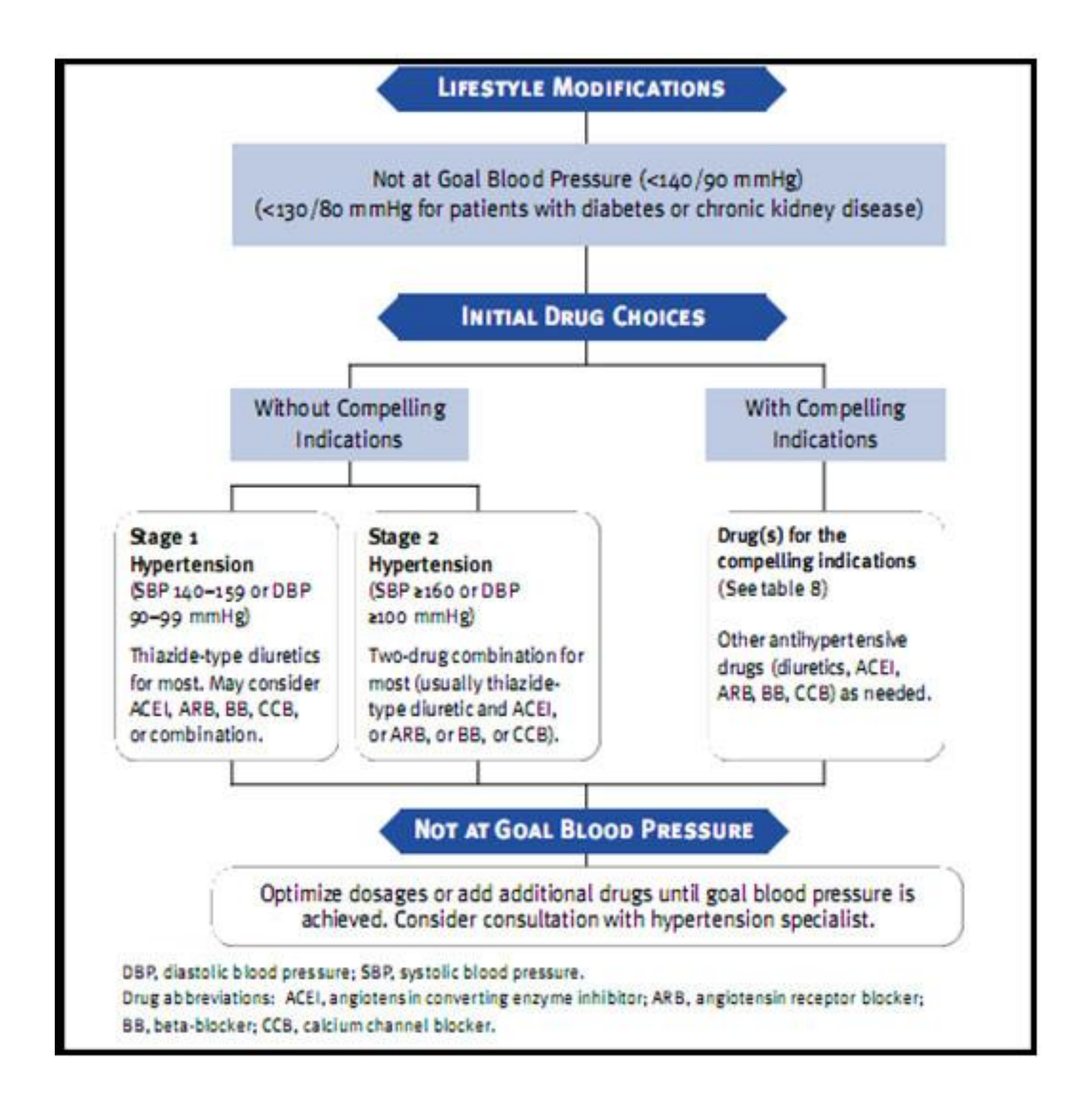
Hypertension and tachycardia from cocaine toxicity rarely require specific treatment. Alpha-adrenergic antagonists (phentolamine) are the preferred agents for cocaine-associated acute coronary syndromes. Pheochromocytoma treatment guidelines are similar to that of cocaine toxicity. Only after alpha blockade can beta blockers be added for BP control.

Preeclampsia/eclampsia

The preferred medications are hydralazine, labetalol, and nifedipine. Avoid - Nitroprusside, angiotensin-converting enzyme inhibitors, esmolol. In women with eclampsia or preeclampsia, the SBP should be < 160 mm Hg and the DBP should be < 110 mm Hg in the antepartum and intrapartum periods. If the platelet count is less than 100,000 cells mm^3, the BP should be maintained below 150/100 mm Hg. Patients with eclampsia or preeclampsia should also be treated with IV magnesium sulfate to avoid seizures.

Perioperative hypertension

Nitroprusside, nitroglycerin, and esmolol are preferred. Target the perioperative BP to within 20% of the patient's baseline pressure, except if there is the potential for life-threatening arterial bleeding. Perioperative beta blockers are the first choice in patients undergoing vascular procedures or in patients with an intermediate or high risk of cardiac complications.


LIFESTYLE MODIFICATIONS
Not at Goal Blood Pressure (<140/90 mmHg)
(<130/80 mmHg for patients with diabetes or chronic kidney disease)
INITIAL DRUG CHOICES
Without Compelling Indications
With Compelling Indications
Stage 1 Hypertension (SBP 140–159 or DBP 90–99 mmHg)
Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB, or combination.
Stage 2 Hypertension (SBP ≥160 or DBP ≥100 mmHg)
Two-drug combination for most (usually thiazide-type diuretic and ACEI, or ARB, or BB, or CCB).
Drug(s) for the compelling indications (See table 8)
Other antihypertensive drugs (diuretics, ACEI, ARB, BB, CCB) as needed.
NOT AT GOAL BLOOD PRESSURE
Optimize dosages or add additional drugs until goal blood pressure is achieved. Consider consultation with hypertension specialist.
DBP, diastolic blood pressure; SBP, systolic blood pressure.
Drug abbreviations: ACEI, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; BB, beta-blocker; CCB, calcium channel blocker.

18.100 THE INFLUENCE OF COMORBIDITY ON THE CHOICE OF ANTIHYPERTENSIVE DRUG THERAPY

Class of drug	Compelling indications	Possible indications	Caution	Compelling contraindications
α-blockers	Benign prostatic hypertrophy	-	Postural hypotension, heart failure[1]	Urinary incontinence
Angiotensin-converting enzyme (ACE) inhibitors	Heart failure	Chronic renal disease[2]	Renal impairment[2]	Pregnancy
	Left ventricular dysfunction, post-myocardial infarction or established coronary heart disease Type 1 diabetic nephropathy Secondary stroke prevention[4]	Type 2 diabetic nephropathy	Peripheral vascular disease[3]	Renovascular disease[2]
Angiotensin II receptor blockers	ACE inhibitor intolerance Type 2 diabetic nephropathy Hypertension with left ventricular hypertrophy Heart failure in ACE-intolerant patients, after myocardial infarction	Left ventricular dysfunction after myocardial infarction Intolerance of other antihypertensive drugs Proteinuric renal disease, chronic renal disease[2] Heart failure	Renal impairment[2] Peripheral vasculardisease[3]	Pregnancy
β-blockers	Myocardial infarction, angina Heart failure[5]		Heart failure[5] Peripheral vascular disease Diabetes (except with coronary heart disease)	Asthma or chronic obstructive pulmonary disease Heart block
Calcium channel blockers (dihydropyridine)	Elderly patients, isolated systolic hypertension	Angina	-	-

DKA

History

Insidious increased thirst (ie, polydipsia) and urination (ie, polyuria) are the most common early symptoms of diabetic ketoacidosis (DKA). Malaise, generalized weakness, and fatigability also can present as symptoms of DKA.

Nausea and vomiting usually occur and may be associated with diffuse abdominal pain, decreased appetite, and anorexia. A history of rapid weight loss is a symptom in patients who are newly diagnosed with type 1 diabetes.

Patients may present with a history of failure to comply with insulin therapy or missed insulin injections due to vomiting or psychological reasons. Decreased perspiration is another possible symptom of DKA.

Altered consciousness in the form of mild disorientation or confusion can occur. Although frank coma is uncommon, it may occur when the condition is neglected or if dehydration or acidosis is severe.

Physical Examination

General signs of diabetic ketoacidosis (DKA) may include the following:

- Ill appearance - Dry skin - Labored respiration - Dry mucous membranes
- Decreased skin turgor - Decreased reflexes - Characteristic acetone (ketotic) breath odor

Effects on vital signs that are related to DKA may include the following:

- Tachycardia - Hypotension - Tachypnea - Hypothermia - Fever, if infection is present

Specific signs of DKA may include the following:

- Confusion - Coma - Abdominal tenderness

The physical examination should also include detection of the signs of possible intercurrent illnesses such as myocardial infarction, urinary tract infection, pneumonia, and perinephric abscess. Search for signs of infection is mandatory in all cases.

Noticing that the body temperature may be within the reference range or low, even in the presence of intercurrent infection, is particularly important.

Signs and Symptoms of Hyperglycemia, Acidosis, and Dehydration

Symptoms of hyperglycemia associated with diabetic ketoacidosis may include thirst, polyuria, polydipsia, and nocturia.

Signs of acidosis may include shallow rapid breathing or air hunger (Kussmaul or sighing respiration), abdominal tenderness, and disturbance of consciousness.

Although these signs are not usual in all cases of diabetic ketoacidosis (DKA), their presence signifies a severe form of DKA.

Signs of dehydration include a weak and rapid pulse, dry tongue and skin, hypotension, and increased capillary refill time.

Complications Associated with DKA

Complications associated with DKA include sepsis and diffuse ischemic processes. Other associated complications include the following:

- CVT - Myocardial infarction - DVT - Acute gastric dilatation
- Erosive gastritis - Late hypoglycemia - Respiratory distress
- Infection (most commonly, urinary tract infections)
- Hypophosphatemia - Mucormycosis - Cerebrovascular accident
- Complicated pregnancy - Trauma - Stress
- Cocaine - Surgery
- Heavy use of concentrated carbohydrate beverages (eg, sodas, sports drinks)
- Acromegaly - Idiopathic condition (20-30%) - Dental abscess

Approach Considerations هام جدا يجب أن تفهموا طريقة العلاج

Managing diabetic ketoacidosis (DKA) in an intensive care unit during the first 24-48 hours always is advisable. When treating patients with DKA, the following points must be considered and closely monitored:

- **Correction of fluid loss with intravenous fluids**
- **Correction of hyperglycemia with insulin**
- **Correction of electrolyte disturbances, particularly potassium loss**
- **Correction of acid-base balance**
- **Treatment of concurrent infection, if present**

It is essential to maintain extreme vigilance for any concomitant process, such as infection, cerebrovascular accident, myocardial infarction, sepsis, or deep venous thrombosis.

It is important to pay close attention to the correction of fluid and electrolyte loss during the first hour of treatment. This always should be followed by gradual correction of hyperglycemia and acidosis. Correction of fluid loss makes the clinical picture clearer and may be sufficient to correct acidosis. The presence of even mild signs of dehydration indicates that at least 3 L of fluid has already been lost.

Patients usually are not discharged from the hospital unless they have been able to switch back to their daily insulin regimen without a recurrence of ketosis. When the condition is stable, pH exceeds 7.3, and bicarbonate is greater than 18 mEq/L, the patient is allowed to eat a meal preceded by a subcutaneous (SC) dose of regular insulin.

Insulin infusion can be discontinued 30 minutes later. If the patient is still nauseated and cannot eat, dextrose infusion should be continued and regular or ultra–short-acting insulin should be administered SC every 4 hours, according to blood glucose level, while trying to maintain blood glucose values at 100-180 mg/dL.

Correction of Fluid Loss

Fluid resuscitation is a critical part of treating patients with DKA. Intravenous solutions replace extravascular and intravascular fluids and electrolyte losses. They also dilute both the glucose level and the levels of circulating counterregulatory hormones. Insulin is needed to help switch from a catabolic state to an anabolic state, with uptake of glucose in tissues and the reduction of gluconeogenesis as well as free fatty acid and ketone production.

Initial correction of fluid loss is either by isotonic sodium chloride solution or by lactated Ringer solution. The recommended schedule for restoring fluids is as follows:

- Administer 1-3 L during the first hour.
- Administer 1 L during the second hour.
- Administer 1 L during the following 2 hours
- Administer 1 L every 4 hours, depending on the degree of dehydration and central venous pressure readings

When the patient becomes euvolemic, the physician may switch to half the isotonic sodium chloride solution, particularly if hypernatremia exists. Isotonic saline should be administered at a rate appropriate to maintain adequate blood pressure and pulse, urinary output, and mental status.

If a patient is severely dehydrated and significant fluid resuscitation is needed, switching to a balanced electrolyte solution (eg, Normosol-R, in which some of the chloride in isotonic saline is replaced with acetate) may help to avoid the development of a hyperchloremic acidosis.

When blood sugar decreases to less than 180 mg/dL, isotonic sodium chloride solution is replaced with 5-10% dextrose with half isotonic sodium chloride solution.

After initial stabilization with isotonic saline, switch to half-normal saline at 200-1000 mL/h (half-normal saline matches losses due to osmotic diuresis).

Insulin should be started about an hour after IV fluid replacement is started to allow for checking potassium levels and because insulin may be more dangerous and less effective before some fluid replacement has been obtained.

Although the incidence of life-threatening hypokalemia due to aggressive insulin administration is very low, there is little to no advantage in starting insulin prior to rehydration and evaluation of serum potassium levels. Initial bolus of insulin does not change overall management of DKA.

Pediatric protocols to minimize the risk of cerebral edema by reducing the rate of fluid repletion vary. The International Society for Pediatric and Adolescent Diabetes (ISPAD) Clinical Practice Consensus Guidelines suggest initial fluid repletion in pediatric patients should be 10-20 mL/kg of normal saline (0.9%) solution during the first 1-2 hours without initial bolus, and then, after 1-2 hours, insulin should be started to avoid pediatric cerebral edema.

ISPAD provides detailed fluid administration guidelines. Total volume over the first 4 hours should not exceed 40-50 mL/kg. Fluid administration is as vital in children as in adults.

Insulin Therapy

When insulin treatment is started in patients with DKA, several points must be considered. A low-dose insulin regimen has the advantage of not inducing the severe hypoglycemia or hypokalemia that may be observed with a high-dose insulin regimen.

Only short-acting insulin is used for correction of hyperglycemia. Subcutaneous absorption of insulin is reduced in DKA because of dehydration; therefore, using intravenous or intramuscular routes is traditionally preferable.

SC use of the fast-acting insulin analog (lispro) has been tried in pediatric DKA (0.15 U/kg q2h). The results were shown to be comparable to IV insulin, but ketosis took 6 additional hours to resolve. Such technically simplified methods may be cost-effective and may preclude admissions to intensive care units in patients with mild cases.

The initial insulin dose is a continuous IV insulin infusion using an infusion pump, if available, at a rate of 0.1 U/kg/h. A mix of 24 units of regular insulin in 60 mL of isotonic sodium chloride solution usually is infused at a rate of 15 mL/h (6 U/h) until the blood glucose level drops to less than 180 mg/dL; the rate of infusion then decreases to 5-7.5 mL/h (2-3 U/h) until the ketoacidotic state abates.

Larger volumes of an insulin and isotonic sodium chloride solution mixture can be used, providing that the infusion dose of insulin is similar. Larger volumes may be easier in the absence of an IV infusion pump (eg, 60 U of insulin in 500 mL of isotonic sodium chloride solution at a rate of 50 mL/h).

The optimal rate of glucose decline is 100 mg/dL/h. Do not allow the blood glucose level to fall below 200 mg/dL during the first 4-5 hours of treatment. Hypoglycemia may develop rapidly with correction of ketoacidosis.

Allowing blood glucose to drop to hypoglycemic levels is a common mistake that usually results in a rebound ketosis derived by counter-regulatory hormones. Rebound ketosis necessitates a longer duration of treatment. The other hazard is that rapid correction of hyperglycemia and hyperosmolarity may shift water rapidly to the hyperosmolar intracellular space and may induce cerebral edema.

Although DKA was a common problem in patients with diabetes who were treated with continuous subcutaneous insulin infusion through insulin infusion pumps, the incidence of DKA was reduced with the introduction of pumps equipped with sensitive electronic alarm systems that alert users when the infusion catheter is blocked.

Electrolyte Correction

If the potassium level is greater than 6 mEq/L, do not administer potassium supplement. If the potassium level is 4.5-6 mEq/L, administer 10 mEq/h of potassium chloride. If the potassium level is 3-4.5 mEq/L, administer 20 mEq/h of potassium chloride.

Monitor serum potassium levels hourly, and the infusion must be stopped if the potassium level is greater than 5 mEq/L. The monitoring of serum potassium must continue even after potassium infusion is stopped in the case of (expected) recurrence of hypokalemia.

In severe hypokalemia, not starting insulin therapy is advisable unless potassium replacement is under way; this is to avert potentially serious cardiac dysrhythmia that may result from hypokalemia.

Potassium replacement should be started with initial fluid replacement if potassium levels are normal or low. Add 20-40 mEq/L of potassium chloride to each liter of fluid once the potassium level is less than 5.5 mEq/L. Potassium can be given as follows: two thirds as KCl, one third as KPO4.

Correction of Acid-Base Balance

Sodium bicarbonate only is infused if decompensated acidosis starts to threaten the patient's life, especially when associated with either sepsis or lactic acidosis. If sodium bicarbonate is indicated, 100-150 mL of 1.4% concentration is infused initially. This may be repeated every half hour if necessary. Rapid and early correction of acidosis with sodium bicarbonate may worsen hypokalemia and cause paradoxical cellular acidosis.

Bicarbonate typically is not replaced as acidosis will improve with the above treatments alone. Administration of bicarbonate has been correlated with cerebral edema in children.

Treatment of Concurrent Infection

In the presence of infection, the administration of proper antibiotics is guided by the results of culture and sensitivity studies. Starting empiric antibiotics on suspicion of infection until culture results are available may be advisable.

Management of Treatment-Related Complications

Cerebral edema

Cerebral edema is a serious, major complication that may evolve at any time during treatment of DKA and primarily affects children. It is the leading cause of DKA mortality in children.

Be extremely cautious to avoid cerebral edema during initiation of therapy. Deterioration of the level of consciousness in spite of improved metabolic state usually indicates the occurrence of cerebral edema. MRI usually is used to confirm the diagnosis.

Cerebral edema that occurs at initiation of therapy tends to worsen during the course of treatment. Mannitol or hypertonic saline should be available if cerebral edema is suspected.

According to Wolfsdorf et al, 0.5-1 g/kg intravenous mannitol may be given over the course of 20 minutes and repeated if no response is seen in 30-120 minutes.[Also, if

no response to mannitol occurs, hypertonic saline (3%) may be given at 5-10 mg/kg over the course of 30 minutes.

Clinical cerebral edema is rare and carries the highest mortality rate. Although mannitol (0.25-1 g/kg IV) and dexamethasone (2-4 mg q6-12h) frequently are used in this situation, no specific medication has proven useful in such instances.

Muir et al have identified diagnostic criteria for cerebral edema that include abnormal response to pain, decorticate and decerebrate posturing, cranial nerve palsies, abnormal central nervous system respiratory patterns, fluctuating level of consciousness, sustained heart rate deceleration, incontinence, and more nonspecific criteria such as vomiting, headache, lethargy, and elevated diastolic blood pressure

Cerebral edema begins with mental status changes and is believed to be due partially to idiogenic osmoles, which have stabilized brain cells from shrinking while the diabetic ketoacidosis was developing.

Cardiac dysrhythmia

Cardiac dysrhythmia may occur secondary to severe hypokalemia and/or acidosis either initially or as a result of therapy in patients with DKA. Usually, correction of the cause is sufficient to treat cardiac dysrhythmia, but if it persists, consultation with a cardiologist is mandatory. Performing cardiac monitoring on patients with DKA during correction of electrolytes always is advisable.

Pulmonary edema

Pulmonary edema may occur for the same reasons as cerebral edema in patients with diabetic ketoacidosis. Be cautious of possible overcorrection of fluid loss, though it occurs only rarely.

Although initial aggressive fluid replacement is necessary in all patients, particular care must be taken in those with comorbidities such as renal failure or congestive heart failure. Diuretics and oxygen therapy often suffice for the management of pulmonary edema.

Myocardial injury

Nonspecific myocardial injury may occur in severe DKA, which is associated with minute elevations of myocardial biomarkers (troponin T and CK-MB) and initial ECG changes compatible with myocardial infarction (MI).

Acidosis and very high levels of free fatty acids could cause membrane instability and biomarker leakage. Coronary arteriography usually is normal, and patients tend to recover fully without further evidence of ischemic heart disease. Regardless of the pathogenesis, the presence of minute biomarker elevations and ECG changes do not necessarily signify MI in DKA.

Hypoglycemia

In patients with diabetic ketoacidosis, hypoglycemia may result from inadequate monitoring of glucose levels during insulin therapy.

Hypokalemia

Hypokalemia is a complication that is precipitated by failing to rapidly address the total body potassium deficit brought out by rehydration and insulin treatment, which not only reduces acidosis but directly facilitates potassium reentry into the cell.

Consultations

An endocrinologist also may be consulted to assist with management after the patient has been stabilized adequately.

Any mental status change in pediatric patients suggests the possibility of cerebral edema, and when this occurs, a pediatric endocrinologist or pediatric intensivist should be consulted as soon as possible. Psychological counseling of young children and adolescents usually is helpful.

Long-Term Monitoring

Frequent blood glucose monitoring at home makes DKA less likely, as this allows them to promptly search for possible reasons for unexpectedly high blood glucose values before the condition progresses to DKA.

In a study of 127 patients with DKA who were admitted to a pediatric intensive care unit, Bradley and Tobias concluded that multiple weaknesses existed in the prehospital care of these patients.[23] These included lack of appropriate laboratory evaluation, excessive insulin dosing (both bolus doses and infusion rates), lack of fluid resuscitation, use of inappropriate fluids for resuscitation, and the use of sodium bicarbonate.

Urinary Calculi

Patients with urinary calculi may report **pain, infection, or hematuria**. The passage of stones into the ureter with subsequent acute obstruction, proximal urinary tract dilation, and spasm is associated with classic renal colic.

Acute onset of severe flank pain radiating to the groin, gross or microscopic hematuria, nausea, and vomiting not associated with an acute abdomen are symptoms that most likely indicate renal colic caused by an acute ureteral or renal pelvic obstruction from a calculus. Renal colic pain rarely, if ever, occurs without obstruction.

Patients with large renal stones known as staghorn calculi (see the image below) are often relatively asymptomatic. The term "staghorn" refers to the presence of a branched kidney stone occupying the renal pelvis and at least one calyceal system. Such calculi usually manifest as infection and hematuria rather than as acute pain.



Complete staghorn calculus that fills the collecting system of the kidney (no intravenous contrast material in this patient). Although many staghorn calculi are struvite (related to infection with urease-splitting bacteria), the density of this stone suggests that it may be metabolic in origin and is likely composed of calcium oxalate. Percutaneous nephrostolithotomy or perhaps even open surgical nephrolithotomy is required to remove this stone.

Asymptomatic bilateral obstruction, which is uncommon, manifests as symptoms of renal failure.

Important historical features are as follows:

- Duration, characteristics, and location of pain
- History of urinary calculi
- Prior complications related to stone manipulation
- Urinary tract infections
- Loss of renal function
- Family history of calculi
- Solitary or transplanted kidney
- Chemical composition of previously passed stones

Location and characteristics of pain

. Location and quality of pain are related to position of the stone within the urinary tract. Severity of pain is related to the degree of obstruction, presence of ureteral spasm, and presence of any associated infection.

Stones obstructing the ureteropelvic junction may present with mild-to-severe deep flank pain without radiation to the groin, due to distention of the renal capsule. Stones impacted within the ureter cause abrupt, severe, colicky pain in the flank and ipsilateral lower abdomen with radiation to the testicles or the vulvar area. Intense nausea, with or without vomiting, usually is present.

Pain from upper ureteral stones tends to radiate to the flank and lumbar areas. On the right side, this can be confused with cholecystitis or cholelithiasis; on the left, the differential diagnoses include acute pancreatitis, peptic ulcer disease, and gastritis.

Midureteral calculi cause pain that radiates anteriorly and caudally. This midureteral pain in particular can easily mimic appendicitis on the right or acute diverticulitis on the left.

Distal ureteral stones cause pain that tends to radiate into the groin or testicle in the male or labia majora in the female because the pain is referred from the ilioinguinal or genitofemoral nerves.

Stones lodged at the ureterovesical junction also may cause irritative voiding symptoms, such as urinary frequency and dysuria. If a stone is lodged in the intramural ureter, symptoms may appear similar to cystitis or urethritis. These symptoms include

suprapubic pain, urinary frequency, urgency, dysuria, stranguria, pain at the tip of the penis, and sometimes various bowel symptoms, such as diarrhea and tenesmus. These symptoms can be confused with pelvic inflammatory disease, ovarian cyst rupture, or torsion and menstrual pain in women.

Calculi that have entered the bladder are usually asymptomatic and are passed relatively easily during urination.

Phases of acute renal colic attack

colic has been described as having 3 clinical phases.

The first phase is the acute or onset phase.The typical attack starts early in the morning or at night, waking the patient from sleep. When it begins during the day, it tends to start slowly and insidiously. The pain is usually steady, increasingly severe, and continuous, sometimes punctuated by intermittent paroxysms of even more excruciating pain.

The second phase is the constant phase. Once the pain reaches maximum intensity, it tends to remain constant until it is either treated or allowed to diminish spontaneously. The period of sustained maximal pain is called the constant phase of the renal colic attack. This phase usually lasts 1-4 hours but can persist longer than 12 hours in some cases. Most patients arrive in the ED during this phase of the attack.

The third phase is the abatement or relief phase.During this final phase, the pain diminishes fairly quickly, and patients finally feel relief.

Other symptoms

Nausea and vomiting occur in at least 50% of patients with acute renal colic. Nausea is caused by the common innervation pathway of the renal pelvis, stomach, and intestines through the celiac axis and vagal nerve afferents.. Nonsteroidal anti-inflammatory drugs (NSAIDs) can often cause gastric irritation and GI upset.

The presence of a renal or ureteral calculus is not a guarantee that the patient does not have some other, unrelated medical problem causing the GI symptoms.

In some cases, a stone may pass before the definitive imaging procedure has been completed. In these cases, residual inflammation and edema still may cause some transient or diminishing obstruction and pain even without any stone being positively identified.

Physical Examination

The classic presentation for a patient with acute renal colic is the sudden onset of severe pain originating in the flank and radiating inferiorly and anteriorly. The pain is usually, but not always, associated with microscopic hematuria, nausea, and vomiting.

Dramatic costovertebral angle tenderness is common; this pain can move to the upper or lower abdominal quadrant as a ureteral stone migrates distally.

Abdominal examination usually is unremarkable. Bowel sounds may be hypoactive, a reflection of mild ileus, which is not uncommon in patients with severe, acute pain.

Peritoneal signs are usually absent—an important consideration in distinguishing renal colic from other sources of flank or abdominal pain. Testicles may be painful but should not be very tender and should appear normal.

Unlike patients with an acute abdomen, who usually try to lie absolutely still, patients with renal colic tend to move constantly, seeking a more comfortable position. (However, patients with pyonephrosis also tend to remain motionless.) The classic patient with renal colic is writhing in pain, pacing about, and unable to lie still, in contrast to a patient with peritoneal irritation, who remains motionless to minimize discomfort.

Lack of hematuria alone does not exclude the diagnosis of acute renal colic. Tachycardia and hypertension are relatively common in these cases, even in patients with no prior personal history of abnormal cardiac or blood pressure problems.

Fever is not part of the presentation of uncomplicated nephrolithiasis. The presence of pyuria, fever, leukocytosis, or bacteriuria suggests the possibility of a urinary infection and the potential for an infected obstructed renal unit or pyonephrosis. Such a condition is potentially life threatening and should be treated as a surgical emergency.

In patients older than 60 years presenting with severe abdominal pain and with no prior history of renal stones, look carefully for physical signs of abdominal aortic aneurysm (AAA)

Complications

Serious complications of urinary tract stone disease include the following:

- Abscess formation - Serious infection of the kidney that diminishes renal function
- Urinary fistula formation - Ureteral scarring and stenosis
- Ureteral perforation - Extravasation - Urosepsis
- Renal loss due to long-standing obstruction

Infected hydronephrosis is the most deadly complication because the presence of infection adjacent to the highly vascular renal parenchyma places the patient at risk for rapidly progressive sepsis and death.

A ureteral stone associated with obstruction and upper UTI is a true urologic emergency.

Complete ureteral obstruction may occur in patients with tightly impacted stones. This is best diagnosed via IVP and is not discernible on noncontrast CT scan. Patients with 2 healthy kidneys can tolerate several days of complete unilateral ureteral obstruction without long-term effects on the obstructed kidney. If a patient with complete obstruction is well hydrated and pain and vomiting are well controlled, the patient can be discharged from the ED with urologic follow-up within 1-2 days.

Differential Diagnoses

- Abdominal Abscess
- Acute Glomerulonephritis
- Appendicitis
- Biliary Colic
- Cholecystitis
- Cholelithiasis
- Diverticulitis
- Duodenal Ulcers
- Epididymitis
- Gastritis and Peptic Ulcer Disease
- Gastrointestinal Foreign Bodies
- Ileus
- Inflammatory Bowel Disease
- Large Bowel Obstruction
- Liver Abscess
- Pancreatitis
- Papillary Necrosis
- Pelvic Inflammatory Disease
- Pyonephrosis
- Rectal Foreign Bodies
- Renal Arteriovenous Malformation
- Renal Cell Carcinoma
- Renal Vein Thrombosis Imaging
- Small Bowel Obstruction
- Splenic Abscess
- Testicular Torsion
- Urinary Tract Infection in Females
- Urinary Tract Infection in Men
- Urinary Tract Obstruction
- Viral Gastroenteritis

Approach Considerations

Acute renal colic with resultant flank pain is a common and sometimes complex clinical problem. Whereas noncontrast abdominopelvic computed tomography (CT) scans have become the imaging modality of choice, in some situations, renal ultrasonography or a contrast study such as intravenous pyelography (IVP) may be preferred.

A kidneys-ureters-bladder (KUB) radiograph, in addition to the renal colic CT scan, facilitates the review and follow-up of stone patients. Alternatively, the “CT scout” (a digital reconstruction from the CT that has an appearance similar to a KUB) is almost as sensitive as a KUB and is a good substitute at the initial assessment if the stone seen on the CT scan is visible on the CT scout

Initial stones in elderly people and in children are relatively uncommon; however, consider kidney stones whenever acute back or flank pain is encountered, regardless of patient age. When stones occur in persons in these uncommon age groups, a metabolic workup consisting of a 24-hour urine collection and appropriate serum laboratory testing is recommended.

Guidelines from the European Association of Urology recommend the following laboratory tests in all patients with an acute stone episode :

- Urinary sediment/dipstick test for demonstration of blood cells, with a test for bacteriuria (nitrite) and urine culture in case of a positive reaction
- Serum creatinine level, as a measure of renal function

In addition, patients with fever warrant a complete blood cell count. Patients with vomiting should have serum or plasma sodium and potassium levels measured.

Urinalysis

Microscopic examination of the urine for evidence of hematuria and infection is a critical part of the evaluation of a patient thought to have renal colic. Gross or

microscopic hematuria is only present in approximately 85% of patients with urinary calculi. The lack of microscopic hematuria does not eliminate renal colic as a potential diagnosis.

Attention should also be paid to the presence or absence of leukocytes, crystals, and bacteria and to the urinary pH. In general, if the number of white blood cells (WBCs) in the urine is greater than 10 cells per high-power field or greater than the number of RBCs, suspect a UTI. Pyuria (>5 WBCs/hpf on a centrifuged specimen) in a patient with ureterolithiasis should prompt a careful search for signs of infected hydronephrosis.

Urinary crystals of calcium oxalate, uric acid, or cystine may occasionally be found upon urinalysis. When present, these crystals are very good clues to the underlying type and nature of any obstructing calculus.

Determining urinary pH also helps. A urine pH greater than 7 suggests presence of urea-splitting organisms, such as ***Proteus, Pseudomonas,*** **or** ***Klebsiella*** **species, and struvite stones. A urine pH less than 5 suggests uric acid stones.**

Blood Studies

Complete blood count

Whereas mild leukocytosis often accompanies a renal colic attack, a high index of suspicion for a possible renal or systemic infection should accompany any serum WBC count of 15,000/µL or higher in a patient presenting with an apparent acute kidney stone attack, even if afebrile. A depressed RBC count suggests a chronic disease state or severe ongoing hematuria.

Serum electrolytes, creatinine, calcium, uric acid, parathyroid hormone, and phosphorus

A high serum uric acid level may indicate gouty diathesis or hyperuricosuria, while hypercalcemia suggests either renal-leak hypercalciuria (with secondary hyperparathyroidism) or primary hyperparathyroidism. If the serum calcium level is elevated, serum PTH levels should be obtained.

24-Hour Urine Profile

To identify urinary risk factors, a 24-hour urine profile, including appropriate serum tests of renal function, uric acid, and calcium, is needed.

The most common findings on 24-hour urine studies include hypercalciuria,hyperoxaluria, hyperuricosuria, hypocitraturia, and low urinary volume. Other factors, such as high urinary sodium and low urinary magnesium concentrations, may also play a role. A finding of hypercalcemia should prompt follow-up with an intact parathyroid hormone study to evaluate for primary and secondary hyperparathyroidism.

Another clinical approach to hypercalciuria, when hyperparathyroidism has been excluded with appropriate blood tests, is avoidance of excessive dietary calcium (usual recommendation, 600-800 mg/d), modest limitation of oxalate intake, and thiazide therapy..

Hyperoxaluria may be primary (a rare genetic disease), enteric. Oxalate restriction and vitamin B-6 supplementation are somewhat helpful in patients with idiopathic hyperoxaluria. Enteric hyperoxaluria is the type that is most amenable to treatment; dietary calcium supplementation often produces dramatic results.

Calcium citrate is the recommended supplement because it tends to further reduce stone formation. Calcium carbonate supplementation is less expensive but lacks citrate's added benefit. Calcium works as an oxalate binder, reducing oxalate absorption from the GI tract. It should be administered with meals, especially those that contain high-oxalate foods. The supplement should not contain added vitamin D, because this increases calcium absorption, leaving less calcium in the GI tract to bind to oxalate. The optimal 24-hour urine oxalate level is 20 mg/d or less.

Hyperuricosuria predisposes to the formation of calcium-containing calculi because sodium urate can produce malabsorption of macromolecular inhibitors or can serve as a nidus for the heterogeneous growth of calcium oxalate crystals. Gouty diathesis, a condition of increased stone production associated with high serum uric acid levels, is also possible.

Therapy involves potassium citrate supplementation, allopurinol, or both. In general, patients with pure uric acid stones and hyperuricemia are treated with allopurinol, and those with hyperuricosuric calcium stones are treated with citrate supplementation. The optimal 24-hour urine uric acid level is 600 mg/d or less.

Sodium and phosphorus

Excess sodium excretion can contribute to hypercalciuria by a phenomenon known as solute drag. Elevated urinary sodium levels are almost always associated with dietary indiscretions. Decreasing the oral sodium intake can decrease calcium excretion, thereby decreasing calcium saturation.

An elevated phosphorus level is useful as a marker for a subtype of absorptive hypercalciuria known as renal phosphate leak (absorptive hypercalciuria type III). Renal phosphate leak is identified by high urinary phosphate levels, low serum phosphate levels, high serum 1,25 vitamin D-3 (calcitriol) levels, and hypercalciuria. This type of hypercalciuria is uncommon and does not respond well to standard therapies.

Citrate and magnesium

Magnesium and, especially, citrate are important chemical inhibitors of stone formation. Hypocitraturia is one of the most common metabolic defects that predispose to stone formation, and some authorities have recommended citrate therapy as primary or adjunctive therapy to almost all patients who have formed recurrent calcium-containing stones.

Lemon juice is an excellent source of citrate; alternatively, large quantities of lemonade can be ingested, and this, of course, has the added benefit of providing increased fluid intake.

Magnesium is a more recently recognized inhibitor of stone formation, and the clinical role of magnesium replacement therapy is less well defined than that of citrate.

pH

Some stones, such as those composed of uric acid or cystine, are pH-dependent, meaning that they can form only in acidic conditions. Calcium phosphate and struvite only form when the urine pH is alkaline. Although the other parameters in the 24-hour urine usually identify patients at risk of forming these stones, pH studies can be important in monitoring these patients, in optimizing therapy with citrate supplementation, and in identifying occult stone disease in some patients.

Plain (Flat Plate or KUB) Radiography

Plain abdominal radiography (also referred to as flat plate or KUB radiography) is useful for assessing total stone burden, as well as the size, shape, composition, and location of urinary calculi in some patients. Calcium-containing stones (approximately 85% of all upper urinary tract calculi) are radiopaque, but pure uric acid, indinavir-induced, and cystine calculi are relatively radiolucent on plain radiography.

When used with other imaging studies, such as a renal ultrasonography or, particularly, CT scanning, the plain film helps provide a better understanding of the characteristics of urinary stones revealed with these other imaging studies. This may also be helpful in planning surgical therapy.

If a stone is not visible on a flat plate radiograph, it could be a radiolucent uric acid stone that can be dissolved with alkalinizing medication. Such a stone is more likely if the urine pH indicates very acidic urine. **In practice, any patient with symptoms of acute renal colic who demonstrates a urine pH lower than 6.0 should be considered at risk for a possible uric acid stone**. If a stone of adequate size is visible on a CT scan but not visible on KUB, then uric stones should be considered.

The flat plate radiograph is inexpensive, quick, and usually helpful even if no specific stone is observed. It is extremely useful in following the progress of previously documented radiopaque calculi and checking the position of any indwelling double-J stents

Ultrasonography

Renal ultrasonography by itself is frequently adequate to determine the presence of a renal stone. The study is mainly used alone in pregnancy or in combination with plain abdominal radiography to determine hydronephrosis or ureteral dilation associated with an abnormal radiographic density believed to be a urinary tract calculus. **A stone easily identified with renal ultrasonography but not visible on the plain radiograph may be a uric acid or cystine stone, which is potentially dissolvable with urinary alkalinization therapy.**

In addition, ultrasonography is not reliable for small stones (ie, those smaller than 5 mm) and does not help in the evaluation of kidney function.

Renal ultrasonography works best in the setting of relatively large stones within the renal pelvis or kidney and sometimes at the UPJ. Whether the stones are radiolucent

or radio-opaque does not matter because an ultrasound image is based strictly on density, not on calcium content. Ultrasonography is a good way to monitor known stones after medical or surgical therapy if the stones are large enough to be detected by this modality and are in a suitable position.

The combination of renal ultrasonography with KUB radiography has been proposed as a reasonable initial evaluation protocol when a CT scan cannot be performed or is unavailable. When combined with KUB radiography, ultrasonography can quickly and inexpensively provide substantial information about the urinary tract without the risk of contrast nephrotoxicity or hypersensitivity. IVP can then be limited to those patients for whom additional information is required for a diagnosis or for whom the etiology of the pain remains unclear.

Future studies may utilize 2-dimensional ultrasonography in combination with color Doppler analysis of the ureteral jets to enhance sensitivity of ultrasonography in patients with ureteral colic.

Intravenous Pyelography (Urography)

Before the advent of helical CT, IVP, also known as intravenous urography (IVU), was the test of choice in diagnosing ureterolithiasis. IVP is widely available and fairly inexpensive but less sensitive than noncontrast helical CT. CT scanning with delayed contrast series and thin slices has reduced the need for IVP in the evaluation of problematic ureteral stones.

The main advantage of IVP is the clear outline of the entire urinary system that it provides, making visualization of even mild hydronephrosis relatively easy. IVP is helpful in identifying the specific problematic stone among numerous pelvic calcifications, as well as in demonstrating renal function and establishing that the other kidney is functional. These determinations are particularly helpful if the degree of hydronephrosis is mild and the noncontrast CT scan findings are not definitive. IVP can also show nonopaque stones as filling defects.

Disadvantages include the need for IV contrast material, which may provoke an allergic response or renal failure, and the need for multiple delayed films, which can take up to 6 hours. Obtaining the IVP is also a relative labor-intensive process. In addition, IVP may fail to reveal alternative pathology if a stone is not discovered, delaying the final diagnosis. False-negative results usually occur with stones located at the ureterovesical junction.

KUB radiographs are obtained immediately before contrast administration and at 1, 5, 10, and 15 minutes afterwards or until visible contrast material fills both ureters (see the image below). Prone films are sometimes obtained to enhance visualization of the ureters. When the bladder is full of contrast and the distal ureters contain sufficient contrast for visualization, the patient is asked to void; then a postvoid film is taken. Sometimes, oblique views are needed when bone or bowel contents overlie the area of interest.



Intravenous pyelogram (IVP) demonstrating dilation of the right renal collecting system and right ureter consistent with right ureterovesical stone.

Look for direct visualization of stone within the ureter, unilateral ureteral dilation, delayed appearance of the nephrogram phase, lack of normal peristalsis pattern of the ureter, or perirenal contrast extravasation. Degree of obstruction is graded based on delay in appearance of the nephrogram.

Typically, an IVP positive for a ureteral stone is one that shows a delayed nephrogram effect and columnization. The ureter is peristaltic, so the entire ureter is not usually visualized on a single film except when an obstruction is present, such as from a stone. Even without observing any specific stone, the presence of a nephrogram effect in one kidney with normal function of the opposite kidney is highly suggestive, but not diagnostic, of ureteral obstruction.

Extravasation of contrast around the collecting system may be a sign of a ruptured fornix, while pyelolymphatic backflow indicates that contrast has entered into the renal lymphatic drainage system. Both are considered signs of a more severe ureteric obstruction.

However, no published study has indicated that the clinical course, treatment outcome, or residual renal damage is altered in any way in these patients. In fact, this information about the radiological assessment of the relative severity of the obstruction rarely affects clinical treatment decisions, except perhaps in persons with solitary kidneys.

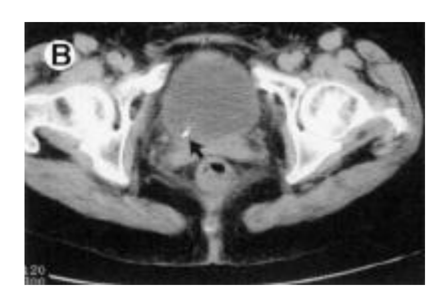
Contrast-induced nephropathy

Contrast-induced nephropathy (CIN) is the third leading cause of hospital-acquired acute renal failure. A serum creatinine level of more than 2 mg/dL is a relative contraindication to the use of IV contrast agents.

Computed Tomography Scanning

At most institutions that offer this examination, CT scanning has replaced IVP, the historic criterion standard, for the assessment of urinary tract stone disease, especially for acute renal colic. CT scans are readily available in most hospitals and can be performed and read in just a few minutes. Numerous studies have demonstrated that CT has a sensitivity of 95-100% and superior specificity and accuracy when compared with IVP.

A renal colic study consists of a noncontrast or unenhanced CT scan of the abdomen and pelvis, including very narrow cuts taken through the kidneys and bladder areas, where symptomatic stones are most likely to be encountered.



Noncontrast helical CT scan of the abdomen demonstrating a stone at the right ureterovesical junction.

An abdominal flat plate or KUB radiograph is sometimes automatically included in a renal colic study, depending on the institution and the preferences of the medical staff.

Advantages of CT scanning include the following:

- It can reveal other pathology (eg, AAAs, appendicitis, pancreatitis, cholecystis, ovarian disorders, diverticular disease, renal carcinoma). If the patient's true underlying pathology is something other than a kidney stone, the CT scan is more clinically useful than an IVP for examining the possibilities.
- It can be performed quickly (< 5 min acquisition time)
- It avoids the use of IV contrast materials.
- The density of the stone can assist in predicting stone composition and response to shockwave lithotripsy.

Disadvantages of CT scanning include the following:

- It is relatively expensive.
- It exposes the patient to a relatively high radiation dose (and thus should not be performed on pregnant women).

Retrograde Pyelography

The most precise imaging method for determining the anatomy of the ureter and renal pelvis and for making a definitive diagnosis of any ureteral calculus is not IVP or renal colic CT scanning but retrograde pyelography.

Nuclear Renal Scanning

A nuclear renal scan can be used to objectively measure differential renal function, especially in a dilated system for which the degree of obstruction is in question. This is also a reasonable study in pregnant patients, in whom radiation exposure must be limited.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) has virtually no role in the current evaluation of acute renal colic in the typical patient. Direct detection of most stones is not possible with MRI, and MRI should not be used for that purpose in most instances. MRIs are generally more expensive than other studies, such as CT scans, which reveal stones much better.

Approach Considerations

Treatment of nephrolithiasis involves emergency management of renal (ureteral) colic, including surgical interventions where indicated, and medical therapy for stone disease.

In emergency settings where concern exists about possible renal failure, **the focus of treatment should be on correcting dehydration, treating urinary infections,**

preventing scarring, identifying patients with a solitary functional kidney, and reducing risks of acute renal failure from contrast nephrotoxicity, particularly in patients with preexisting azotemia (creatinine >2 mg/dL), diabetes, dehydration, or multiple myeloma. Adequate intravenous (IV) hydration is essential to minimize the nephrotoxic effects of IV contrast agents. Choosing imaging studies that do not require IV contrast (eg, ultrasonography, plain abdominal flat plate radiographs, noncontrast computed tomography [CT] scans) is wise, especially in patients at increased risk for developing renal failure.

Most small stones with relatively mild hydronephrosis can be treated with observation and acetaminophen. More serious cases with intractable pain may require drainage with a stent or percutaneous nephrostomy. The internal ureteral stent is usually preferred in these situations because of decreased morbidity.

Acetaminophen can be used in pregnancy for mild-to-moderate pain.

A stone chemical composition analysis should be performed whenever possible, and information should be provided to motivated patients about possible 24-hour urine testing for long-term nephrolithiasis prophylaxis

The size of the stone is an important predictor of spontaneous passage**. A stone less than 4 mm in diameter has an 80% chance of spontaneous passage**; this falls to 20% for stones larger than 8 mm in diameter. **However, stone passage also depends on the exact shape and location of the stone and the specific anatomy of the upper urinary tract in the particular individual**. For example, the presence of a ureteropelvic junction (UPJ) obstruction or a ureteral stricture could make passing even very small stones difficult or impossible. Most experienced emergency department (ED) physicians and urologists have observed very large stones passing and some very small stones that do not move.

Aggressive medical therapy has shown promise in increasing the spontaneous stone passage rate and relieving discomfort while minimizing narcotic usage. **Aggressive treatment of any proximal urinary infection is important to avoid potentially dangerous pyonephrosis and urosepsis.** In these cases, consider percutaneous nephrostomy drainage rather than retrograde endoscopy, especially in very ill patients.

Medical therapy for stone disease takes both short- and long-term forms. The former includes measures to dissolve the stone (possible only with noncalcium stones)

or to facilitate stone passage, and the latter includes treatment to prevent further stone formation. Stone prevention should be considered most strongly in patients who have risk factors for increased stone activity, including stone formation before age 30 years, family history of stones, multiple stones at presentation, and residual stones after surgical treatment.

Indications for hospitalization

The decision to hospitalize a patient with a stone is usually made based on clinical grounds rather than on any specific finding on a radiograph. Generally, hospitalization for an acute renal colic attack is now officially termed an observation because most

patients recover sufficiently to go home within 24 hours. Admission rate for patients with acute renal colic is approximately 20%.

Hospital admission is clearly necessary when any of the following is present:

- Oral analgesics are insufficient to manage the pain.
- Ureteral obstruction from a stone occurs in a solitary or transplanted kidney.
- Ureteral obstruction from a stone occurs in the presence of a urinary tract infection (UTI), fever, sepsis, or pyonephrosis.

Infected hydronephrosis, defined as UTI proximal to an obstructing stone, mandates hospital admission for antibiotics and prompt drainage. Midstream urine culture and sensitivity was a poor predictor of infected hydronephrosis in one series, being positive in only 30% of cases.[31]

The clinical presentation of infected hydronephrosis is variable. Pyuria (>5 white blood cells [WBCs] per high-power field [hpf]) is almost always present but is not diagnostic of proximal infection

Antibiotics should cover *Escherichia coli* and *Staphylococcus, Enterobacter, Proteus,* and *Klebsiella* species. In another small study of 38 patients with hydronephrosis, 16 had infected hydronephrosis and 22 had sterile hydronephrosis. Ultrasonography alone detected 6 of 16 cases of pyonephrosis, a sensitivity of 38%. Using a cutoff value of 3 mg/dL for C-reactive protein and 100 mm/h for erythrocyte sedimentation rate, the diagnostic accuracy of detecting infected hydronephrosis and pyonephrosis increased to 97%.

Relative indications to consider for a possible admission include comorbid conditions (eg, diabetes), dehydration requiring prolonged intravenous (IV) fluid therapy, renal failure, or any immunocompromised state. Patients with complete obstruction, perinephric urine extravasation, a solitary kidney, or pregnancy, and those with a poor social support system, also should be considered for admission, especially if rapid urologic follow-up is not reliably available.

Larger stones (ie, ≥ 7 mm) that are unlikely to pass spontaneously require some type of surgical procedure. In some cases, hospitalizing a patient with a large stone to facilitate surgical stone intervention is reasonable. However, most patients with acute renal colic can be treated on an ambulatory basis.

About 15-20% of patients require invasive intervention due to stone size, continued obstruction, infection, or intractable pain. Several techniques are available to the urologist when the stone fails to pass spontaneously, including extracorporeal shock wave lithotripsy (SWL), ureteroscopy, and percutaneous nephrolithotomy.

Emergency Management of Renal Colic

Initial treatment of a renal colic patient in the ED **starts with obtaining IV access to allow fluid, analgesic, and antiemetic medications to be administered**. Many of these patients are dehydrated from poor oral intake and vomiting. Although the role of supranormal hydration in the management of renal (ureteral) colic is controversial (see

below), patients who are dehydrated or ill need adequate restoration of circulating volume.

After diagnosing renal (ureteral) colic, determine the presence or absence of obstruction or infection. Obstruction in the absence of infection can be initially managed with analgesics and with other medical measures to facilitate passage of the stone. Infection in the absence of obstruction can be initially managed with antimicrobial therapy. In either case, promptly refer the patient to a urologist.

If neither obstruction nor infection is present, analgesics and other medical measures to facilitate passage of the stone (see below) can be initiated with the expectation that the stone will likely pass from the upper urinary tract if its diameter is smaller than 5-6 mm (larger stones are more likely to require surgical measures).

If both obstruction and infection are present, emergency decompression of the upper urinary collecting system is required (see Surgical Care). In addition, immediately consult with a urologist for patients whose pain fails to respond to ED management.

Pain relief

The cornerstone of ureteral colic management is analgesia, which can be achieved most expediently with parenteral narcotics or **nonsteroidal anti-inflammatory drugs (NSAIDs**).

Parenteral narcotics are the mainstay of analgesia for patients with acute renal colic. They work primarily on the central nervous system (CNS) to reduce the perception of pain. They are inexpensive and quite effective. **When considering a medication and dosage range, remember that acute renal colic is probably the most painful malady to affect humans**. Adverse effects of narcotic analgesics include respiratory depression, sedation, constipation, a potential for addiction, nausea, and vomiting.

Morphine is a potent narcotic analgesic that controls severe pain primarily through a CNS mechanism via specific receptor site interactions.

Adverse effects of morphine include respiratory depression, drowsiness, mood changes, nausea, vomiting, increases in the cerebrospinal fluid pressure, and cough reflex depression. The most bothersome is respiratory depression caused by a direct effect on the brain stem respiratory center. This effect is most severe in patients who are elderly, debilitated, or both.

Naloxone (0.4 mg or 1 mL) is a specific narcotic antagonist for both meperidine and morphine sulfate that can be administered to counteract inadvertent narcotic overdosage or unusual opioid sensitivity. Naloxone has no analgesic properties.

Of the NSAIDs, **the only one approved by the US Food and Drug Administration (FDA) for parenteral use is ketorolac. Ketorolac works at the peripheral site of pain production rather than on the CNS**. It has been proven in multiple studies to be as effective as opioid analgesics, with fewer adverse effects. The dosage is 30-60 mg IM or 30 mg IV initially followed by 30 mg IV or IM every 6-8 hours. A dose of 15 mg is recommended in patients older than 65 years.

Some practitioners use parenteral ketorolac in the hospital but recommend either ibuprofen or oral cyclooxygenase-2 inhibitors (eg, celecoxib or meloxicam) for pain management in outpatients.

A maximum of 5 days of ketorolac therapy is recommended.

Chemically, ketorolac is similar to aspirin and may increase the prothrombin time when administered with anticoagulants. Ketorolac can increase methotrexate toxicity and phenytoin levels. It is potentiated by probenecid and should be avoided in patients with peptic ulcer disease, renal failure, or recent gastrointestinal (GI) bleeding.

Antiemetic therapy

Metoclopramide is the only antiemetic that has been specifically studied in the treatment of renal colic. In 2 double-blinded studies, it apparently provided pain relief equivalent to narcotic analgesics in addition to relieving nausea. Its antiemetic effect stems from its dopaminergic receptor blockage in the CNS. It has no anxiolytic activity and is less sedating than other centrally acting dopamine antagonists. The effect of metoclopramide begins within 3 minutes of an IV injection, but it may not take effect for as long as 15 minutes if administered IM.

The usual dose in adults is 10 mg IV or IM every 4-6 hours as needed. Metoclopramide is not available as a suppository.

Antidiuretic therapy

Several studies have now demonstrated that **desmopressin** (DDAVP), a potent antidiuretic that is essentially an antidiuretic hormone, can dramatically reduce the pain of acute renal colic in many patients. It acts quickly, has no apparent adverse effects, reduces the need for supplemental analgesic medications, and may be the only immediate therapy necessary for some patients. It is available as a nasal spray (usual dose of 40 mcg, with 10 mcg per spray) and as an IV injection (4 mcg/mL, with 1 mL the usual dose). Generally, only 1 dose is administered.

Although desmopressin is thought to work by reducing the intraureteral pressure, it may also have some direct relaxing effect on the renal pelvic and ureteral musculature.

While some of the human studies lack adequate controls and further studies must be conducted, desmopressin therapy currently appears to be a promising alternative or adjunct to analgesic medications in patients with acute renal colic, especially in patients in whom narcotics cannot be used or in whom the pain is unusually resistant to standard medical treatment.

Antibiotic therapy

Use antibiotics if a kidney stone or ureteral obstruction has been diagnosed and the patient has clinical evidence of a UTI. Evidence of a possible UTI includes an abnormal finding upon microscopic urinalysis, showing pyuria of 10 WBCs/hpf (or more WBCs than RBCs), bacteriuria, fever, or unexplained leukocytosis. Perform a urine culture in these cases because a culture cannot be performed reliably later should the infection prove resistant to the prescribed antibiotic.

Active medical expulsive therapy

The traditional outpatient treatment approach detailed above has recently been improved with the application of a more aggressive treatment approach known as active medical expulsive therapy (MET). Many randomized trials have confirmed the efficacy of MET in reducing the pain of stone passage, increasing the frequency of stone passage, and reducing the need for surgery.

MET should be considered in any patient with a reasonable probability of stone passage. Given that stones smaller than 3 mm are already associated with an 85% chance of spontaneous passage, MET is probably most useful for stones 3-10 mm in size. Overall, MET is associated with a 65% greater likelihood of stone passage.

The original rationale for MET was based on the possible causes of failure to spontaneously pass a stone, **including ureteral stricture, muscle spasm, local edema, inflammation, and infection**. Various common drugs were considered that would potentially benefit these problems, improve spontaneous stone passage, and alleviate renal colic discomfort.

Although NSAIDs have ureteral-relaxing effects and, as such, can be considered a form of MET, patient outcomes have been significantly improved only with the use of more potent (off-label) medications. The initially popularized regimens for MET included corticosteroids such as prednisone, as in the following example:

- Ketorolac at 10 mg orally every 6 hours for 5 days
- Nifedipine XL at 30 mg/d PO for 7 days
- Prednisone 20 mg PO twice a day for 5 days
- Trimethoprim/sulfamethoxazole DS once a day for 7 days
- Acetaminophen 2 tablets 4 times a day for 7 days
- An oral opioid pain medication (oxycodone-acetaminophen) as needed for breakthrough pain
- Prochlorperazine suppository as needed for control of nausea

Although corticosteroids are effective, concerns about their side effects (admittedly not supported by randomized data) limited the acceptance of MET. More recently, randomized studies have demonstrated great efficacy of the following individual agents, sparing the corticosteroid component.

The calcium channel blocker nifedipine is indicated for angina, migraine headaches, Raynaud disease, and hypertension, but it can also reduce muscle spasms in the ureter, which helps reduce pain and facilitate stone passage. Ureteral smooth muscle uses an active calcium pump to produce contractions, so a calcium channel blocker such as nifedipine would be expected to relax ureteral muscle spasms.

The alpha-blockers, such as terazosin, and the alpha-1 selective blockers, such as tamsulosin, also relax the musculature of the ureter and lower urinary tract, markedly facilitating passage of ureteral stones. Some literature suggests that the alpha-blockers are more effective in this setting than the calcium channel blockers, and most practitioners currently use alpha-blockers preferentially over calcium channel blockers.

Multiple prospective randomized controlled studies in the urology literature have demonstrated that patients treated with oral alpha-blockers have an increased rate of spontaneous stone passage and a decreased time to stone passage. **The best studied of these is tamsulosin, 0.4 mg administered daily.**

A systematic review by Singh et al found that MET using either alpha antagonists or calcium channel blockers augmented the stone expulsion rate for moderately sized distal ureteral stones. Adverse effects were noted in 4% of those taking alpha antagonists and in 15.2% of those taking calcium channel blockers.

MET with calcium channel blockers and alpha-blockers also appears to improve the results of ESWL (see Surgical Care) inasmuch as the stone fragments resulting from treatment appear to clear the system more effectively.

- A typical regimen for this aggressive therapy is as follows:
- 1-2 oral acetaminophen tablets every 4 hours as needed for pain
- 600-800 mg ibuprofen every 8 hours

MET with 30 mg nifedipine extended-release tablet once daily, 0.4 mg tamsulosin once daily, or 4 mg of terazosin once daily

Limit MET to a 10- to 14-day course, as most stones that pass during this regimen do so in that time frame. If outpatient treatment fails, promptly consult a urologist.

Intravenous hydration

IV hydration in the setting of acute renal colic is controversial. Whereas some authorities believe that IV fluids hasten passage of the stone through the urogenital system, others express concern that additional hydrostatic pressure exacerbates the pain of renal colic. One small study of 43 ED patients found no difference in pain score or rate of stone passage in patients who received 2 L of saline over 2 hours versus those who received 20 mL of saline per hour.

IV hydration should be given to patients with clinical signs of dehydration or to those with a borderline serum creatinine level who must undergo intravenous pyelography (IVP).

Straining urine for stones

Collecting any passed kidney stones is extremely important in the evaluation of a patient with nephrolithiasis for stone-preventive therapy. Yet, in a busy ED, the simple instruction to strain all the urine for stones can be easily overlooked.

Surgical Care

In general, stones that are 4 mm in diameter or smaller will probably pass spontaneously, and stones that are larger than 8 mm are unlikely to pass without surgical intervention.

With MET, stones 5-8 mm in size often pass, especially if located in the distal ureter. The larger the stone, the lower the possibility of spontaneous passage (and thus the

greater the possibility that surgery will be required), although many other factors determine what happens with a particular stone.

Indications and contraindications

The primary indications for surgical treatment include **pain, infection, and obstruction.** Infection combined with urinary tract obstruction is an extremely dangerous situation, with significant risk of urosepsis and death, and must be treated emergently in virtually all cases. Additionally, certain occupational and health-related reasons exist.

General contraindications to definitive stone manipulation include the following:

- Active, untreated UTI
- Uncorrected bleeding diathesis
- Pregnancy (a relative, but not absolute, contraindication)

Specific contraindications may apply to a given treatment modality. For example, do not perform SWL if a ureteral obstruction is distal to the calculus or in pregnancy.

Surgical options

For an obstructed and infected collecting system secondary to stone disease, virtually no contraindications exist for emergency surgical relief either by ureteral stent placement (a small tube placed endoscopically into the entire length of the ureter from the kidney to the bladder) or by percutaneous nephrostomy (a small tube placed through the skin of the flank directly into the kidney).

Many urologists have a preference for one technique or the other, but, in general, patients who are acutely ill, who have significant medical comorbidities, or who harbor stones that probably cannot be bypassed with ureteral stents undergo percutaneous nephrostomy, while others receive ureteral stent placement.

The vast majority of symptomatic urinary tract calculi are now treated with noninvasive or minimally invasive techniques, while open surgical excision of a stone from the urinary tract is now limited to isolated atypical cases.

Guidelines are now available to assist the urologist in selecting surgical treatments. The 2005 American Urological Association (AUA) staghorn calculus guidelines recommend percutaneous nephrostolithotomy as the cornerstone of management.In the ureteral stone guidelines produced by a joint effort of the AUA and the European Association of Urology, SWL and ureteroscopy are both recognized as first-line treatments for ureteral stones

Stent placement

Internal ureteral stents form a coil at either end when the stiffening insertion guide wire is removed. One coil forms in the renal pelvis and the other in the bladder.

The major drawback of stents, however, is that they are often quite uncomfortable for patients due to direct bladder irritation, spasm, and reflux. This discomfort can be alleviated to some extent by pain medications, anticholinergics (eg, oxybutynin, tolterodine), alpha-blockers, and topical analgesics (eg, phenazopyridine).

Percutaneous nephrostomy

In some cases, drainage of an obstructed kidney is necessary and stent placement is inadvisable or impossible. In particular, such cases include patients with pyonephrosis who have a UTI or urosepsis exacerbated by an obstructing calculus. In these patients, retrograde endourological procedures like retrograde pyelography and stent placement may exacerbate infection by pushing infected urinary material into the obstructed renal unit. Percutaneous nephrostomy is useful in such situations.

Extracorporeal shockwave lithotripsy

ESWL, the least invasive of the surgical methods of stone removal, utilizes an underwater energy wave focused on the stone to shatter it into passable fragments.

It is especially suitable for stones that are smaller than 2 cm and lodged in the upper or middle calyx.

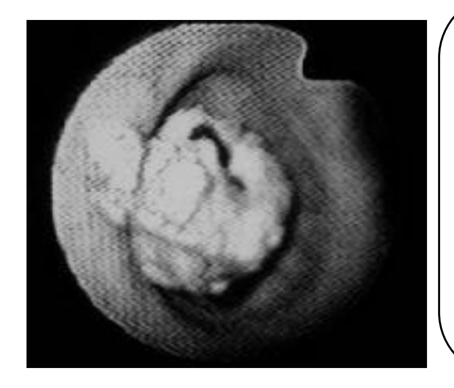
It is contraindicated in pregnancy, untreatable bleeding disorders, tightly impacted stones, or in cases of ureteral obstruction distal to the stone. In addition, the effectiveness is limited for very hard stones (which tend to be dense on CT scan), cystine stones, and in very large patients.

The patient, under varying degrees of anesthesia (depending on the type of lithotriptor used), is placed on a table or in a gantry that is then brought into contact with the shock head. The deeper the anesthesia (general endotracheal), the better the results.

SWL is limited somewhat by the size and location of the calculus. A stone larger than 1.5 cm in diameter or one located in the lower section of the kidney is treated less successfully. Fragmentation still occurs, but the large volume of fragments or their location in a dependent section of the kidney precludes complete passage.

Ureteroscopy

Along with SWL, ureteroscopic manipulation of a stone (see the image below) is a commonly applied method of stone removal. A small endoscope, which may be rigid, semirigid, or flexible, is passed into the bladder and up the ureter to directly visualize the stone.



Two calculi in a dependent calyx of the kidney (lower pole) visualized through a flexible fiberoptic ureteroscope. In another location, these calculi might have been treated with extracorporeal shockwave lithotripsy (ESWL), but, after being counseled regarding the lower success rate of ESWL for stones in a dependent location, the patient elected ureteroscopy. Note that the image provided by fiberoptics, although still acceptable, is inferior to that provided by the rod-lens optics of the rigid ureteroscope in the previous picture.

Ureteroscopy is especially suitable for removal of stones that are 1-2 cm, lodged in the lower calyx or below, cystine stones, and high attenuation ("hard") stones. The typical patient has acute symptoms caused by a distal ureteral stone, usually measuring 5-8 mm. Stones smaller than 5 mm in diameter generally are retrieved

using a stone basket, whereas tightly impacted stones or those larger than 5 mm are manipulated proximally for SWL or are fragmented using an endoscopic direct-contact fragmentation device.

Often, a ureteral stent must be placed after ureteroscopy in order to prevent obstruction from ureteral spasm and edema. Since a ureteral stent is often uncomfortable, many urologists eschew stent placement following ureteroscopy in selected patients.

Percutaneous nephrostolithotomy

Percutaneous nephrostolithotomy allows fragmentation and removal of large calculi from the kidney and ureter. Percutaneous nephrostolithotomy is especially useful for stones larger than 2 cm in diameter.

In some cases, a combination of SWL and a percutaneous technique is necessary to completely remove all stone material from a kidney. This technique, called sandwich therapy, is reserved for staghorn or other complicated stone cases. In such cases, experience has shown that the final procedure should be percutaneous nephrostolithotomy.

Open nephrostomy

Medical Therapy for Stone Disease

Dissolution of calculi

Urinary calculi composed predominantly of calcium cannot be dissolved with current medical therapy; however, medical therapy is important in the long-term chemoprophylaxis of further calculus growth or formation.

Uric acid and cystine calculi can be dissolved with medical therapy. Patients with uric acid stones who do not require urgent surgical intervention for reasons of pain, obstruction, or infection can often have their stones dissolved with **alkalization of the urine**. Sodium bicarbonate can be used as the alkalizing agent, but **potassium citrate** is usually preferred because of the availability of slow-release tablets and the avoidance of a high sodium load.

The dosage of the alkalizing agent should be adjusted to maintain the urinary pH between 6.5 and 7.0. Urinary pH of more than 7.5 should be avoided because of the potential deposition of calcium phosphate around the uric acid calculus, which would make it undissolvable. Both uric acid and cystine calculi form in acidic environments.

Even very large uric acid calculi can be dissolved in patients who comply with therapy. Roughly 1 cm per month dissolution can be achieved. Practical ability to alkalinize the urine significantly limits the ability to dissolve cystine calculi.

Chemoprophylaxis

Prophylactic therapy might include limitation of dietary components, addition of stone-formation inhibitors or intestinal calcium binders, and, most importantly, augmentation of fluid intake.

Besides advising patients to avoid excessive salt and protein intake and to increase fluid intake, base medical therapy for long-term chemoprophylaxis of urinary calculi on the results of a 24-hour urinalysis for chemical constituents.

Chemoprophylaxis of uric acid and cystine calculi consists primarily of long-term alkalinization of urine. If hyperuricosuria or hyperuricemia is documented in patients with pure uric acid stones (present in only a relative minority), allopurinol (300 mg qd) is recommended because it reduces uric acid excretion.

Pharmaceuticals that can bind free cystine in the urine (eg, D-penicillamine, 2-alpha-mercaptopropionyl-glycine) help reduce stone formation in cystinuria. Therapy should also include long-term urinary alkalinization and aggressive fluid intake. Captopril has been shown to be effective in some trials, although, again, strong data are lacking. Routine use should be avoided but can be added in patients who have difficulty in dissolving and preventing cystine stones.

Dietary Measures

In almost all patients in whom stones form, **an increase in fluid intake** and, therefore, an increase in urine output is recommended. This is likely the single most important aspect of stone prophylaxis. Patients with recurrent nephrolithiasis traditionally have been instructed to **drink 8 glasses of fluid daily to maintain adequate hydration and decrease chance of urinary supersaturation with stone-forming salts**. The goal is a total urine volume in 24 hours in excess of 2 liters.

The only other general dietary guidelines are to **avoid excessive salt and protein intake**. Moderation of calcium and oxalate intake is also reasonable.

Dietary calcium should not be restricted beyond normal unless specifically indicated based on 24-hour urinalysis findings. Urinary calcium levels are normal in many patients with calcium stones**. Reducing dietary calcium in these patients may actually worsen their stone disease, because more oxalate is absorbed from the GI tract in the absence of sufficient intestinal calcium to bind with it. This results in a net increase in oxalate absorption and hyperoxaluria**, which tends to increase new kidney stone formation in patients with calcium oxalate calculi.

As a rule, dietary calcium should be restricted to 600-800 mg/d in patients with diet-responsive hypercalciuria who form calcium stones. This is roughly equivalent to a single high-calcium or dairy meal per day.

Prevention of Nephrolithiasis

The most common causes of kidney stones are hypercalciuria, hyperuricosuria, hyperoxaluria, hypocitraturia, and low urinary volume. Each of these major factors can be measured easily with a 24-hour urine sample using one of several commercial

laboratory packages now available. Kidney stone preventive therapy consists of dietary adjustments, nutritional supplements, medications, or combinations of these.

Strongly encourage patients who have a stone at a young age (ie, < 25 y), multiple recurrences, a solitary functioning kidney, or a history of prior kidney stone surgery to obtain a 24-hour urine collection for stone prevention analysis, especially if they are motivated to comply with a long-term stone prevention program. These 24-hour urine collection kits can be obtained from a number of commercial medical laboratories.

Treatment of Primary Hyperoxaluria

Patients with primary hyperoxaluria usually present with a urinary oxalate level in excess of 100 mg/d. Early medical treatment is required to decrease the oxalate level and to prevent deterioration of renal function.

Dietary oxalate restrictions are of no substantial benefit in this type of hyperoxaluric disease. Several medications have been useful.

New and future treatment modalities under investigation include probiotic supplementation, chaperones and hepatocyte cell transplantation, and recombinant gene therapy to replace the enzyme.

High-dose pyridoxine (vitamin B-6)

Pyridoxine deficiency is known to increase urinary oxalate excretion. High-dose pyridoxine may reduce the production of oxalate by enhancing the conversion of glyoxylate to glycine, thereby reducing the substrate available for metabolism to oxalate. A daily dose of 150-500 mg may be required to sufficiently reduce the oxalate level. A normal urinary oxalate level (< 40 mg/d) can be achieved in some patients.

Magnesium

Supplementation with magnesium in the form of magnesium hydroxide and magnesium oxide has been used. Magnesium can complex with oxalate in the intestinal tract, reducing the level of available free oxalate and urinary calcium oxalate supersaturation. It does not directly affect the increased endogenous production of oxalate

When used in combination with pyridoxine, significant reductions in urinary oxalate levels have been noted.

Increased urinary volume

It is essential to increase urinary volume. Optimal 24-hour urinary volumes of 3-4 L/d may be needed to ameliorate the effects of severe hyperoxaluria. Increasing urine volume usually requires multiple nightly disruptions of sleep for extra water consumption.

Thiazides have been shown to decrease urinary oxalate excretion somewhat, possibly by decreasing intestinal oxalate transport.

Other factors that can contribute to stone formation, such as urinary citrate and uric acid, need to be optimized.

IntestinalObstruction

Pathophysiology

Small-bowel obstruction (SBO) leads to proximal dilatation of the intestine due to accumulation of GI secretions and swallowed air. This bowel dilatation stimulates cell secretory activity, resulting in more fluid accumulation. This leads to increased peristalsis above and below the obstruction, with frequent loose stools and flatus early in its course.

Vomiting occurs if the level of obstruction is proximal. Increasing small-bowel distention leads to increased intraluminal pressures. This can cause compression of mucosal lymphatics, leading to bowel wall lymphedema.

With even higher intraluminal hydrostatic pressures, increased hydrostatic pressure in the capillary beds results in massive third spacing of fluid, electrolytes, and proteins into the intestinal lumen. **The fluid loss and dehydration that ensue may be severe and contribute to increased morbidity and mortality**.

Bacteria in the gut proliferate proximal to the obstruction. Microvascular changes in the bowel wall allow translocation to the mesenteric lymph nodes. This is associated with an increase in the incidence of bacteremia due to *Escherichia coli,* but the clinical significance is unclear.

Etiology

The most common cause of small-bowel obstruction (SBO) is postsurgical adhesions

Prevention of SBO may be essentially limited to decreasing the risk of adhesion formation by decreasing the number of intra-abdominal procedures (ie, laparotomies) and resultant scar formation.

Another commonly identified cause of SBO is an incarcerated groin hernia. Other etiologies include malignant tumor (20%), hernia (10%), inflammatory bowel disease (5%), volvulus (3%), and miscellaneous causes (2%). The causes of SBO in pediatric patients include congenital atresia, pyloric stenosis, and intussusception.

Prognosis

Complications of SBO include the following:

- Sepsis - Intra-abdominal abscess - Wound dehiscence - Aspiration
- Short-bowel syndrome (as a result of multiple surgeries)
- Death (secondary to delayed treatment)

History

Obstruction can be characterized as either partial or complete versus simple or strangulated. No accurate clinical picture exists to detect early strangulation of obstruction.

Abdominal pain, often described as crampy and intermittent, is more prevalent in simple obstruction. Often, the presentation may provide clues to the approximate location and nature of the obstruction.

Usually, pain that occurs for a shorter duration of time and is colicky and accompanied by bilious vomiting may be more proximal. Pain that lasts as long as several days, is progressive in nature, and is accompanied by abdominal distention may be typical of a more distal obstruction.

Changes in the character of the pain may indicate the development of a more serious complication (ie, constant pain of a strangulated or ischemic bowel).

Patients also report the following:

- Nausea
- Vomiting - Associated more with proximal obstructions
- Diarrhea - An early finding
- Constipation - A late finding, as evidenced by the absence of flatus or bowel movements
- Fever and tachycardia - Occur late and may be associated with strangulation
- Previous abdominal or pelvic surgery, previous radiation therapy, or both - May be part of the patient's medical history
- History of malignancy - Particularly ovarian and colonic malignancy

Physical Examination

Abdominal distention is present. The duodenal or proximal small bowel has less distention when obstructed than the distal bowel has when obstructed. Hyperactive bowel sounds occur early as GI contents attempt to overcome the obstruction; hypoactive bowel sounds occur late.

Exclude incarcerated hernias of the groin, femoral triangle, and obturator foramina. Proper genitourinary and pelvic examinations are essential.

Look for the following during rectal examination:

- Gross or occult blood, which suggests late strangulation or malignancy
- Masses, which suggest obturator hernia

Check for symptoms commonly believed to be more diagnostic of intestinal ischemia, including the following:

- Fever (temperature >100°F)
- Tachycardia (>100 beats/min)
- Peritoneal signs

No reliable way exists to differentiate simple from early strangulated obstruction on physical examination. Serial abdominal examinations are important and may detect changes early.

Diagnostic Considerations

The following conditions should be considered in the differential diagnosis

- Esophageal rupture or tear
- GI foreign body
- Gastroenteritis

- Inflammatory bowel disease
- Mesenteric ischemia
- Large-bowel obstruction
- Ovarian torsion
- Pancreatitis
- Acute appendicitis
- Diabetic ketoacidosis
- Gastroenteritis
- Intussusception
- Pelvic inflammatory disease
- Urinary Tract Infection

Differential Diagnoses

- Abortion, Threatened
- Cholecystitis and Biliary Colic in Emergency Medicine
- Cholelithiasis
- Constipation
- Diverticular Disease
- Dysmenorrhea
- Endometriosis
- Inflammatory Bowel Disease
- Mesenteric Ischemia

Approach Considerations

If the diagnosis is unclear, admission and observation are warranted to detect early obstructions. Essential laboratory tests are needed; these include the following:

- Blood urea nitrogen (BUN) level - If the BUN level is increased, this may indicate decreased volume state (eg, dehydration)
- Creatinine level - Creatinine level elevations may indicate dehydration
- Complete blood count (CBC) - The white blood cell (WBC) count may be elevated with a left shift in simple or strangulated obstructions; increased hematocrit is an indicator of volume state (ie, dehydration)
- Lactate dehydrogenase tests
- Urinalysis
- Type and crossmatch - The patient may require surgical intervention
- Laboratory tests to exclude biliary or hepatic disease are also needed; they include the following:
- Phosphate level
- Creatine kinase level
- Liver panels

Plain Radiography

Plain radiographs are diagnostically more accurate in cases of simple obstruction. However, diagnostic failure rates of as much as 30% have been reported.

In one small study, the sensitivity of plain radiography was reported to be 75%, and specificity was reported to be 53%; similar findings were reported in a second study. In another study, plain films were more accurate in the detection of acute SBO and the accuracy was higher if interpreted by more-experienced radiologists.

Dilated small-bowel loops with air-fluid levels indicate SBO, as does absent or minimal colonic gas. SBO is demonstrated in the radiographs below.



Small bowel obstruction..

CT Scanning and MRI

CT scanning

Computed tomography (CT) scanning is the study of choice if the patient has fever, tachycardia, localized abdominal pain, and/or leukocytosis.

CT scanning is useful in making an early diagnosis of strangulated obstruction and in delineating the myriad other causes of acute abdominal pain, particularly when clinical and radiographic findings are inconclusive. It also has proved useful in distinguishing the etiologies of small-bowel obstruction (SBO), that is, in distinguishing extrinsic causes (such as adhesions and hernia) from intrinsic causes (such as neoplasms and Crohn disease). In addition, CT scanning differentiates the above from intraluminal causes, such as bezoars. The modality may be less useful in the evaluation of small bowel ischemia associated with obstruction.

CT scanning is capable of revealing abscess, inflammatory process, extraluminal pathology resulting in obstruction, and mesenteric ischemia and enables the clinician to distinguish between ileus and mechanical small bowel obstruction in postoperative patients.

The modality does not require oral contrast for the diagnosis of SBO, because the retained intraluminal fluid serves as a natural contrast agent.

MRI

The accuracy of magnetic resonance imaging (MRI) almost approaches that of CT scanning for the detection of obstructions. MRI is also effective in defining the location and etiology of obstruction.

Ultrasonography

Ultrasonography is less costly and invasive than CT scanning and may reliably exclude SBO in as many as 89% of patients; specificity is reportedly 100%.

Approach Considerations

Emergency Department Care

Initial emergency department (ED) treatment consists of **aggressive fluid resuscitation**,

bowel decompression, administration of analgesia and antiemetic as indicated clinically, early surgical consultation, and administration of antibiotics. (Antibiotics are used to cover against gram-negative and anaerobic organisms.)

Initial decompression can be performed by placement of a nasogastric (NG) tube for suctioning GI contents and preventing aspiration. Monitor airway, breathing, and circulation (ABCs).

Blood pressure monitoring, as well as cardiac monitoring in selected patients (especially elderly patients or those with comorbid conditions), is important.

Nonoperative inpatient care

Continued NG suction provides symptomatic relief, decreases the need for intraoperative decompression, and benefits all patients. No clinical advantage to using a long tube (nasointestinal) instead of a short tube (NG) has been observed.

A nonoperative trial of as many as 3 days is warranted for partial or simple obstruction. Provide adequate fluid resuscitation and NG suctioning. Resolution of obstruction occurs in virtually all patients with these lesions within 72 hours.

Nonoperative treatment for several types of SBO are as follows:

- Malignant tumor - Obstruction by tumor is usually caused by metastasis; initial treatment should be nonoperative (surgical resection is recommended when feasible)
- Inflammatory bowel disease - To reduce the inflammatory process, treatment generally is nonoperative in combination with high-dose steroids; consider parenteral treatment for prolonged periods of bowel rest, and undertake surgical treatment, bowel resection, and/or stricturoplasty if nonoperative treatment fails.
- Intra-abdominal abscess - CT scan–guided drainage is usually sufficient to relieve obstruction
- Radiation enteritis - If obstruction follows radiation therapy acutely, nonoperative treatment accompanied by steroids is usually sufficient; if the obstruction is a chronic sequela of radiation therapy, surgical treatment is indicated
- Incarcerated hernia - Initially use manual reduction and observation; advise elective hernia repair as soon as possible after reduction
- Acute postoperative obstruction - This is difficult to diagnose, because symptoms often are attributed to incisional pain and postoperative ileus; treatment should be nonoperative
- Adhesions - Decreasing intraoperative trauma to the peritoneal surfaces can prevent adhesion formation

Surgical Care

A strangulated obstruction is a surgical emergency. In patients with a complete small-bowel obstruction (SBO), the risk of strangulation is high and early surgical intervention is warranted. Patients with simple complete obstructions in whom nonoperative trials fail also need surgical treatment but experience no apparent disadvantage to delayed surgery.

Laparoscopy has been shown to be safe and effective in selected cases of SBO. A review of retrospective clinical trials showed that laparoscopy showed better results in terms of hospital stay and mortality reduction versus open surgery, but prospective, randomized, controlled trials to assess all outcomes are still needed.

Medication Summary

Fluid replacement with aggressive intravenous (IV) resuscitation using isotonic saline or lactated Ringer solution is indicated.

Oxygen and appropriate monitoring are also required. **Antibiotics** are used to cover gram-negative and anaerobic organisms. In addition, analgesia and antiemetic are

administered as indicated clinically. As previously mentioned, a nonoperative trial of as many as 3 days is warranted for partial or simple obstruction. Resolution of obstruction occurs in virtually all patients with these lesions within 72 hours.

Appendicitis

Background

Appendicitis is defined as an inflammation of the inner lining of the vermiform appendix that spreads to its other parts

In fact, despite diagnostic and therapeutic advancement in medicine, appendicitis remains a clinical emergency and is one of the more common causes of acute abdominal pain.

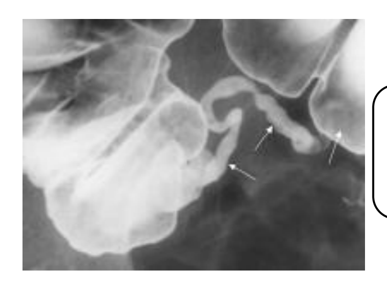
No single sign, symptom, or diagnostic test accurately confirms the diagnosis of appendiceal inflammation in all cases, and the classic history of anorexia and periumbilical pain followed by nausea, right lower quadrant (RLQ) pain, and vomiting occurs in only 50% of cases (see Clinical Presentation).

Appendicitis may occur for several reasons, such as an infection of the appendix, but the most important factor is the obstruction of the appendiceal lumen . Left untreated, appendicitis has the potential for severe complications, including perforation or sepsis, and may even cause death (see Prognosis and Complications). However, the differential diagnosis of appendicitis is often a clinical challenge because appendicitis can mimic several abdominal conditions

Appendectomy remains the only curative treatment of appendicitis .

Anatomy

The appendix is a wormlike extension of the cecum and, for this reason, has been called the vermiform appendix. The average length of the appendix is 8-10 cm (ranging from 2-20 cm). The appendix appears during the fifth month of gestation, and several lymphoid follicles are scattered in its mucosa. Such follicles increase in number when individuals are aged 8-20 years. A normal appendix is seen below.



Normal appendix; barium enema radiographic examination. A complete contrast-filled appendix is observed (arrows), which effectively excludes the diagnosis of appendicitis.

The appendix is contained within the visceral peritoneum that forms the serosa, and its exterior layer is longitudinal and derived from the taenia coli; the deeper, interior muscle layer is circular. Beneath these layers lies the submucosal layer, which contains lymphoepithelial tissue.

Taenia coli converge on the posteromedial area of the cecum, which is the site of the appendiceal base. The appendix runs into a serosal sheet of the peritoneum called the mesoappendix, within which courses the appendicular artery, which is derived from the ileocolic artery. Sometimes, an accessory appendicular artery (deriving from the posterior cecal artery) may be found.

Appendiceal vasculature

The vasculature of the appendix must be addressed to avoid intraoperative hemorrhages. The appendicular artery is contained within the mesenteric fold that arises from a peritoneal extension from the terminal ileum to the medial aspect of the cecum and appendix; it is a terminal branch of the ileocolic artery and runs adjacent to the appendicular wall. Venous drainage is via the ileocolic veins and the right colic vein into the portal vein; lymphatic drainage occurs via the ileocolic nodes along the course of the superior mesenteric artery to the celiac nodes and cisterna chyli.

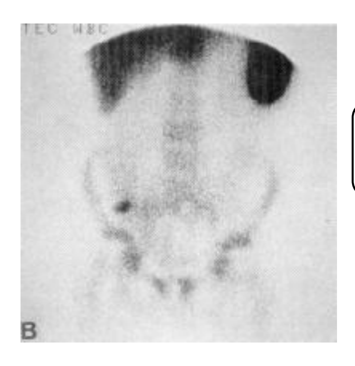
Appendiceal location

The appendix has no fixed position. It originates 1.7-2.5 cm below the terminal ileum, either in a dorsomedial location (most common) from the cecal fundus, directly beside the ileal orifice, or as a funnel-shaped opening (2-3% of patients). The appendix has a retroperitoneal location in 65% of patients and may descend into the iliac fossa in 31%.

In fact, many individuals may have an appendix located in the retroperitoneal space; in the pelvis; or behind the terminal ileum, cecum, ascending colon, or liver. Thus, the course of the appendix, the position of its tip, and the difference in appendiceal position considerably changes clinical findings, accounting for the nonspecific signs and symptoms of appendicitis.

Pathophysiology

Reportedly, appendicitis is caused by obstruction of the appendiceal lumen from a variety of causes (see Etiology). Independent of the etiology, obstruction is believed to cause an increase in pressure within the lumen. Such an increase is related to continuous secretion of fluids and mucus from the mucosa and the stagnation of this material. At the same time, intestinal bacteria within the appendix multiply, leading to the recruitment of white blood cells (see the image below) and the formation of pus and subsequent higher intraluminal pressure.



Technetium-99m radionuclide scan of the abdomen shows focal uptake of labeled WBCs in the right lower quadrant consistent with acute appendicitis.

If appendiceal obstruction persists, intraluminal pressure rises ultimately above that of the appendiceal veins, leading to venous outflow obstruction. As a consequence, appendiceal wall ischemia begins, resulting in a loss of epithelial integrity and allowing bacterial invasion of the appendiceal wall.

Within a few hours, this localized condition may worsen because of thrombosis of the appendicular artery and veins, leading to perforation and gangrene of the appendix. As this process continues, a periappendicular abscess or peritonitismay occur.

Etiology

Appendicitis is caused by obstruction of the appendiceal lumen. The most common causes of luminal obstruction include lymphoid hyperplasia secondary to inflammatory bowel disease (IBD) or infections (more common during childhood and in young adults), fecal stasis and fecaliths (more common in elderly patients), parasites (especially in Eastern countries), or, more rarely, foreign bodies and neoplasms.

History

Variations in the position of the appendix, age of the patient, and degree of inflammation make the clinical presentation of appendicitis notoriously inconsistent. Statistics report that 1 of 5 cases of appendicitis is misdiagnosed; however, a normal appendix is found in 15-40% of patients who have an emergency appendectomy.

Niwa et al reported an interesting case of a young woman with recurrent pain in who was referred for appendicitis, treated with antibiotics, and was found to have an appendiceal diverticulitis associated with a rare pelvic pseudocyst at laparotomy after 12 months. Her condition was probably due to diverticular perforation of the pseudocyst.

Symptoms

The classic history of anorexia and periumbilical pain followed by nausea, right lower quadrant (RLQ) pain, and vomiting occurs in only 50% of cases.

Nausea is present in 61-92% of patients; anorexia is present in 74-78% of patients.

In addition, when vomiting occurs, it nearly always follows the onset of pain.

Vomiting that precedes pain is suggestive of intestinal obstruction, and the diagnosis of appendicitis should be reconsidered. Diarrhea or constipation is noted in as many as 18% of patients and should not be used to discard the possibility of appendicitis.

The most common symptom of appendicitis is abdominal pain.

Typically, symptoms begin as periumbilical or epigastric pain migrating to the right lower quadrant (RLQ) of the abdomen. **This pain migration is the most discriminating feature of the patient's history**, .

Patients usually lie down, flex their hips, and draw their knees up to reduce movements and to avoid worsening their pain. Later, a worsening progressive pain along with vomiting, nausea, and anorexia are described by the patient. Usually, a fever is not present at this stage.

In addition to recording the history of the abdominal pain, obtain a complete summary of the recent personal history surrounding gastroenterologic, genitourinary, and pneumologic conditions, as well as consider gynecologic history in female patients.

An inflamed appendix near the urinary bladder or ureter can cause irritative voiding symptoms and hematuria or pyuria.

Consider the possibility of an inflamed pelvic appendix in male patients with apparent cystitis. **Also consider the possibility of appendicitis in pediatric or adult patients who present with acute urinary retention**

Physical Examination

It is important to remember that the position of the appendix is variable. the base of the appendix was located at the McBurney point in only 4% of patients; in 36%, the base was within 3 cm of the point; in 28%, it was 3-5 cm from that point; and, in 36% of patients, the base of the appendix was more than 5 cm from the McBurney point.

The most specific physical findings in appendicitis are rebound tenderness, pain on percussion, rigidity, and guarding. Although RLQ tenderness is present in 96% of patients, this is a nonspecific finding. Rarely, left lower quadrant (LLQ) tenderness has been the major manifestation in patients with situs inversus or in patients with a lengthy appendix that extends into the LLQ. **Tenderness on palpation in the RLQ over the McBurney point is the most important sign in these patients.**

A careful physical examination, not limited to the abdomen, must be performed in any patient with suspected appendicitis. Gastrointestinal (GI), genitourinary, and pulmonary systems must be studied. Male infants and children occasionally present with an inflamed hemiscrotum due to migration of an inflamed appendix or pus through a patent processus vaginalis. This is often initially misdiagnosed as acute testicular torsion.

In addition, perform a rectal examination in any patient with an unclear clinical picture, and perform a pelvic examination in all women with abdominal pain.

According to the American College of Emergency Physicians (ACEP) 2010 clinical policy update, clinical signs and symptoms should be used to stratify patient risk and to choose next steps for testing and management.

Accessory signs

In a minority of patients with acute appendicitis, some other signs may be noted. However, their absence never should be used to rule out appendiceal inflammation.

The Rovsing sign (RLQ pain with palpation of the LLQ) suggests peritoneal irritation in the RLQ precipitated by palpation at a remote location.

The obturator sign (RLQ pain with internal and external rotation of the flexed right hip) suggests that the inflamed appendix is located deep in the right hemipelvis.

The psoas sign (RLQ pain with extension of the right hip or with flexion of the right hip against resistance) suggests that an inflamed appendix is located along the course of the right psoas muscle.

The Dunphy sign (sharp pain in the RLQ elicited by a voluntary cough) may be helpful in making the clinical diagnosis of localized peritonitis.

Similarly, RLQ pain in response to percussion of a remote quadrant of the abdomen, or to firm percussion of the patient's heel, suggests peritoneal inflammation.

The Markle sign, pain elicited in a certain area of the abdomen when the standing patient drops from standing on toes to the heels with a jarring landing.

Rectal examination

There is no evidence in the medical literature that the digital rectal examination (DRE) provides useful information in the evaluation of patients with suspected appendicitis; however, failure to perform a rectal examination is frequently cited in successful malpractice claims. In 2008, Sedlak et al studied 577 patients who underwent DRE as part of an evaluation for suspected appendicitis and found no value as a means of distinguishing patients with and without appendicitis.

Appendicitis and Pregnancy

The incidence of appendicitis is unchanged in pregnancy relative to the general population, but the clinical presentation is more variable than at other times.

During pregnancy, the appendix migrates in a counterclockwise direction toward the right kidney, rising above the iliac crest at about 4.5 months' gestation. **RLQ pain and tenderness dominate in the first trimester, but in the latter half of pregnancy, right upper quadrant (RUQ) or right flank pain must be considered a possible sign of appendiceal inflammation.**

Nausea, vomiting, and anorexia are common in uncomplicated first trimester pregnancies, but their reappearance later in gestation should be viewed with suspicion.

Diagnostic Scoring

Several investigators have created diagnostic scoring systems to predict the likelihood of acute appendicitis. In these systems, a finite number of clinical variables is elicited from the patient and each is given a numeric value; then, the sum of these values is used.

The best known of these scoring systems is the MANTRELS score, which tabulates migration of pain, anorexia, nausea and/or vomiting, tenderness in the RLQ, rebound tenderness, elevated temperature, leukocytosis, and shift to the left (see Table 1). Table 1. MANTRELS Score

Characteristic	Score
M = Migration of pain to the RLQ	1
A = Anorexia	1
N = Nausea and vomiting	1
T = Tenderness in RLQ	2
R = Rebound pain	1
E = Elevated temperature	1

L = Leukocytosis	2
S = Shift of WBCs to the left	1
Total	10
Source: Alvarado.	
RLQ = right lower quadrant; WBCs = white blood cells	

Clinical scoring systems are attractive because of their simplicity; however, none has been shown prospectively to improve on the clinician's judgment in the subset of patients evaluated in the emergency department (ED) for abdominal pain suggestive of appendicitis. The MANTRELS score, in fact, was based on a population of patients hospitalized for suspected appendicitis, which differs markedly from the population seen in the ED.

Scoring systems and computer-aided diagnosis

Computer-aided diagnosis consists of using retrospective data of clinical features of patients with appendicitis and other causes of abdominal pain and then prospectively assessing the risk of appendicitis. Computer-aided diagnosis can achieve a sensitivity greater than 90% while reducing rates of perforation and negative laparotomy by as much as 50%.

However, the principle disadvantages to this method are that each institution must generate its own database to reflect characteristics of its local population, and specialized equipment and significant initiation time are required. In addition, computer-aided diagnosis is not widely available in US EDs.

Stages of Appendicitis

The stages of appendicitis can be divided into early, suppurative, gangrenous, perforated, phlegmonous, spontaneous resolving, recurrent, and chronic.

Early stage appendicitis

In the early stage of appendicitis, obstruction of the appendiceal lumen leads to mucosal edema, mucosal ulceration, bacterial diapedesis, appendiceal distention due to accumulated fluid, and increasing intraluminal pressure. The visceral afferent nerve fibers are stimulated, and the patient perceives mild visceral periumbilical or epigastric pain, which usually lasts 4-6 hours.

Suppurative appendicitis

Increasing intraluminal pressures eventually exceed capillary perfusion pressure, which is associated with obstructed lymphatic and venous drainage and allows bacterial and inflammatory fluid invasion of the tense appendiceal wall. Transmural spread of bacteria causes acute suppurative appendicitis. When the inflamed serosa of the appendix comes in contact with the parietal peritoneum, patients typically experience the classic shift of pain from the periumbilicus to the right lower abdominal quadrant (RLQ), which is continuous and more severe than the early visceral pain.

Gangrenous appendicitis

Intramural venous and arterial thromboses ensue, resulting in gangrenous appendicitis.

Perforated appendicitis

Persisting tissue ischemia results in appendiceal infarction and perforation. Perforation can cause localized or generalized peritonitis.

Phlegmonous appendicitis or abscess

An inflamed or perforated appendix can be walled off by the adjacent greater omentum or small-bowel loops, resulting in phlegmonous appendicitis or focal abscess.

Spontaneously resolving appendicitis

If the obstruction of the appendiceal lumen is relieved, acute appendicitis may resolve spontaneously. This occurs if the cause of the symptoms is lymphoid hyperplasia or when a fecalith is expelled from the lumen.

Recurrent appendicitis

The incidence of recurrent appendicitis is 10%. The diagnosis is accepted as such if the patient underwent similar occurrences of RLQ pain at different times that, after appendectomy, were histopathologically proven to be the result of an inflamed appendix.

Chronic appendicitis

Chronic appendicitis occurs with an incidence of 1% and is defined by the following: (1) the patient has a history of RLQ pain of at least 3 weeks' duration without an alternative diagnosis; (2) after appendectomy, the patient experiences complete relief of symptoms; (3) histopathologically, the symptoms were proven to be the result of chronic active inflammation of the appendiceal wall or fibrosis of the appendix.

The classic history of anorexia and periumbilical pain followed by nausea, right lower quadrant (RLQ) pain, and vomiting occurs in only 50% of cases. Vomiting that precedes pain is suggestive of intestinal obstruction, and the diagnosis of appendicitis should be reconsidered.

The differential diagnosis of appendicitis is often a clinical challenge because appendicitis can mimic several abdominal conditions (see Differentials). Patients with many other disorders present with symptoms similar to those of appendicitis, such as the following:

- Pelvic inflammatory disease (PID) or tubo-ovarian abscess
- Endometriosis - Ovarian cyst or torsion - Ureterolithiasis and renal colic
- Degenerating uterine leiomyomata - Diverticulitis
- Crohn disease - Colonic carcinoma - Rectus sheath hematoma
- Cholecystitis - Bacterial enteritis - Mesenteric adenitis and ischemia

- Omental torsion
- Urinary tract infection (UTI)
- Pancreatitis
- Biliary colic
- Gastroenteritis
- Perforated duodenal ulcer
- Renal colic
- Enterocolitis

Other problems that should be considered in a patient with suspected appendicitis include appendiceal stump appendicitis, typhlitis, epiploic appendagitis, psoas abscess, and yersiniosis.

Misdiagnosis in women of childbearing age

Appendicitis is misdiagnosed in 33% of nonpregnant women of childbearing age. The most frequent misdiagnoses are PID, followed by gastroenteritis and urinary tract infection.

In distinguishing appendiceal pain from that of PID, anorexia and onset of pain more than 14 days after menses suggests appendicitis.

Previous PID, vaginal discharge, or urinary symptoms indicates PID. On physical examination, tenderness outside the RLQ, cervical motion tenderness, vaginal discharge, and positive urinalysis support the diagnosis of PID.

Although negative appendectomy does not appear to adversely affect maternal or fetal health, diagnostic delay with perforation does increase fetal and maternal morbidity. Therefore, aggressive evaluation of the appendix is warranted in pregnant women.

The level of urinary beta–human chorionic gonadotropin (beta-hCG) is useful in differentiating appendicitis from early ectopic pregnancy. However, with regard to the WBC count, physiologic leukocytosis during pregnancy makes this study less useful in the diagnosis than at other times, and no reliable distinguishing WBC parameters are cited in the literature.

Misdiagnosis in children

Appendicitis is misdiagnosed in 25-30% of children, and the rate of initial misdiagnosis is inversely related to the age of the patient. The most common misdiagnosis is gastroenteritis, followed by upper respiratory infection and lower respiratory infection.

Children with misdiagnosed appendicitis are more likely than their counterparts to have vomiting before pain onset, diarrhea, constipation, dysuria, signs and symptoms of upper respiratory infection, and lethargy or irritability. Physical findings less likely to be documented in children with a misdiagnosis than in others include bowel sounds; peritoneal signs; rectal findings; and ear, nose, and throat findings.

Differential Diagnoses

تعرف عن كل موضوع بأهم ما يميزه في سطر واحد حتي تستطيع التفرقة والاستبعاد

- Abdominal Abscess
- Cholecystitis and Biliary Colic
- Constipation
- Crohn Disease
- Diverticular Disease
- Ectopic Pregnancy
- Endometriosis
- Gastroenteritis, Bacterial
- Gastroenteritis
- Inflammatory Bowel Disease

- Meckel Diverticulum
- Mesenteric Lymphadenitis
- Ovarian Cysts
- Pediatrics, Intussusception
- Renal Calculi
- Urinary Tract Infection
- Mesenteric Ischemia
- Omental Torsion
- Ovarian Torsion
- Pelvic Inflammatory Disease
- Spider Envenomations, Widow

Approach Considerations

Patients with appendicitis may not have the reported classic clinical picture 37-45% of the time, especially when the appendix is located in an unusual place .

In such cases, imaging studies may be important but not always available. However, patients with appendicitis usually have accessory signs that may be helpful for .For example, the obturator sign is present when the internal rotation of the thigh elicits pain (ie, pelvic appendicitis), and the psoas sign is present when the extension of the right thigh elicits pain (ie, retroperitoneal or retrocecal appendicitis).

Laboratory tests are not specific for appendicitis, but they may be helpful to confirm diagnosis in patients with an atypical presentation.

CBC Count

Studies consistently show that 80-85% of adults with appendicitis have a white blood cell (WBC) count greater than 10,500 cells/µL. Neutrophilia greater than 75% occurs in 78% of patients. Less than 4% of patients with appendicitis have a WBC count less than 10,500 cells/µL and neutrophilia less than 75%.

CBC tests are inexpensive, rapid, and widely available; however, the findings are nonspecific. In infants and elderly patients, a WBC count is especially unreliable because these patients may not mount a normal response to infection. In pregnant women, the physiologic leukocytosis renders the CBC count useless for the diagnosis of appendicitis.

C-Reactive Protein

C-reactive protein (CRP) is an acute-phase reactant synthesized by the liver in response to infection or inflammation and rapidly increases within the first 12 hours. CRP has been reported to be useful in the diagnosis of appendicitis; however, it lacks specificity and cannot be used to distinguish between sites of infection.

CRP levels of greater than 1 mg/dL are commonly reported in patients with appendicitis, but very high levels of CRP in patients with appendicitis indicate gangrenous evolution of the disease, especially if it is associated with leukocytosis and neutrophilia. However, CRP normalization occurs 12 hours after onset of symptoms. Several prospective studies have shown that, in adults who have had symptoms for longer than 24 hours, a normal CRP level has a negative predictive value of 97-100% for appendicitis. Thimsen et al noted that a normal CRP level after 12 hours of symptoms was 100% predictive of benign, self-limited illness.

CRP sensitivity

Multiple studies have examined the sensitivity of CRP level alone for the diagnosis of appendicitis in patients selected to undergo appendectomy

Sensitivity of WBC count and CRP level in combination

Investigators have also studied the ability of combinations of WBC count and CRP to reliably rule out the diagnosis of appendicitis.

Triple screen of WBC count, CRP level, and neutrophilia

Mohammed et al prospectively studied 216 children admitted for suspected appendicitis and found a triple screen sensitivity of 86% and a negative predictive value of 81. However, Yang et al found that only 6 of 740 patients with appendicitis had a WBC count less than 10,500 cells/μ L AND neutrophilia that was less than 75%, AND a normal CRP level, yielding a sensitivity of 99.2% for the "triple screen."

Liver and Pancreatic Function Tests

Liver and pancreatic function tests (eg, transaminases, bilirubin, alkaline phosphatase, serum lipase, amylase) may be helpful to determine the diagnosis in patients with an unclear presentation.

Urinalysis اقرأوا هذا الكلام بعناية

Urinalysis may be useful in differentiating appendicitis from urinary tract conditions. Mild pyuria may occur in patients with appendicitis because of the relationship of the appendix with the right ureter. Severe pyuria is a more common finding in urinary tract infections (UTIs). Proteinuria and hematuria suggest genitourinary diseases or hemocoagulative disorders.

One study of 500 patients with acute appendicitis revealed that approximately one third reported urinary symptoms, most commonly dysuria or right flank pain. One in 7 patients had pyuria greater than 10 WBCs per high power field (hpf), and 1 in 6 patients had greater than 3 red blood cells (RBCs) per hpf. Thus, the diagnosis of appendicitis should not be dismissed due to the presence of urologic symptoms or abnormal urinalysis.

Urinary Beta-HCG فحص يجب أن تجريه لكل سيدة

وفي الدول الغربية يجرونه حتي لكل بنت بسبب العلاقات الغير شرعية

For women of childbearing age, the level of urinary beta–human chorionic gonadotropin (beta-hCG) is useful in differentiating appendicitis from early ectopic pregnancy.

Urinary 5-HIAA

According to a report by Bolandparvaz et al, measurement of the urinary 5-hydroxyindoleacetic acid (U-5-HIAA) levels could be an early marker of appendicitis.The rationale of such measurement is related to the large amount of serotonin-secreting cells in the appendix. The investigators noted that U-5-HIAA levels increased significantly in acute appendicitis, decreasing when the inflammation shifted

to necrosis of the appendix. Therefore, such decrease could be an early warning sign of perforation of the appendix.

CT Scanning

Computed tomography (CT) scanning with oral contrast medium or rectal Gastrografin enema has become the most important imaging study in the evaluation of patients with atypical presentations of appendicitis. Intravenous contrast is usually not necessary.

Studies have found a decrease in negative laparotomy rate and appendiceal perforation rate when pelvic CT imaging was used in selected patients with suspected appendicitis. An enlarged appendix is shown in the CT below.



CT scan reveals an enlarged appendix with thickened walls, which do not fill with colonic contrast agent, lying adjacent to the right psoas muscle.

The use of CT has dramatically increased since the introduction of multidetector CT (MDCT) scanners. A large, single center study found that MDCT has a high rate of sensitivity and specificity (98.5% and 98%, respectively) for diagnosing acute appendicitis.

In adults with appendicitis, the diagnostic performance of CT scans with intravenous contrast alone is comparable to that of scans with both intravenous and oral contrast, and patients who receive CT scans with intravenous contrast alone are discharged more quickly from the emergency department.

. Ultrasonography may offer a safer alternative as a primary diagnostic tool for appendicitis, with CT scanning used in those cases in which ultrasonograms are negative or inconclusive.

Ultrasonography

Because of concerns about patient exposure to radiation during CT scans, ultrasonography has been suggested as a safer primary diagnostic modality for appendicitis, with CT scanning used secondarily when ultrasonograms are negative or inconclusive.

In pediatric patients, the ACEP 2010 clinical policy update recommends using ultrasonography for confirmation, but not exclusion, of acute appendicitis. To definitively exclude acute appendicitis, CT is recommended.

A healthy appendix usually cannot be viewed with ultrasonography. When appendicitis occurs, the ultrasonogram typically demonstrates a noncompressible tubular structure of 7-9 mm in diameter (see the images below).



Sagittal graded compression transabdominal sonogram shows an acutely inflamed appendix. The tubular structure is noncompressible, lacks peristalsis, and measures greater than 6 mm in diameter. A thin rim of periappendiceal fluid is present.



Transverse graded compression transabdominal sonogram of an acutely inflamed appendix. Note the targetlike appearance due to thickened wall and surrounding loculated fluid collection.

Vaginal ultrasonography alone or in combination with transabdominal scan may be useful to determine the diagnosis in women of childbearing age. One study of 22 pregnant women in the first and second trimesters showed that graded compression ultrasonography had a sensitivity of 66% and specificity of 95%.

Abdominal Radiography

The kidneys-ureters-bladder (KUB) radiographic view is typically used to visualize an appendicolith in a patient with symptoms consistent with appendicitis (see the following image). This finding is highly suggestive of appendicitis, but appendicoliths also occur in fewer than 10% of cases. The consensus in the literature is that plain radiographs are insensitive, nonspecific, and not cost-effective.

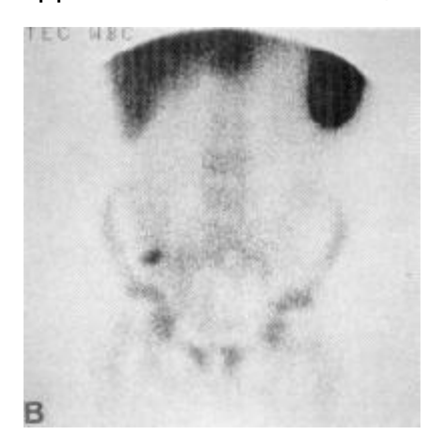


Kidneys-ureters-bladder (KUB) radiograph shows an appendicolith in the right lower quadrant. An appendicolith is seen in fewer than 10% of patients with appendicitis, but, when present, it is essentially pathognomonic.

Radionuclide Scanning

Whole blood is withdrawn for radionuclide scanning. Neutrophils and macrophages are labeled with technetium Tc 99m (^{99m} Tc) albumin and administered intravenously. Then, images of the abdomen

and pelvis are obtained serially over 4 hours. Localized uptake of tracer in the RLQ suggests appendiceal inflammation; this is shown in the images below.



Technetium-99m radionuclide scan of the abdomen shows focal uptake of labeled WBCs in the right lower quadrant consistent with acute appendicitis.

MRI

Magnetic resonance imaging (MRI) plays a relatively limited role in the evaluation of appendicitis because of its high cost, long scan times, and limited availability. However, the lack of ionizing radiation makes it an attractive modality in pregnant patients. In fact, Cobben et al showed that MRI is far superior to transabdominal ultrasonography in evaluating pregnant patients with suspected appendicitis.

Nonetheless, when evaluating pregnant patients with suspected appendicitis, graded compression ultrasonography should be the imaging test of choice.

If ultrasonography demonstrates an inflamed appendix, the patient should undergo appendectomy. If graded compression ultrasonography is nondiagnostic, the patient should undergo MRI of the abdomen and pelvis.

Approach Considerations

Appendectomy remains the only curative treatment of appendicitis, but management of patients with an appendiceal mass can usually be divided into the following 3 treatment categories:

- Patients with a phlegmon or a small abscess: After intravenous (IV) antibiotic therapy, an interval appendectomy can be performed 4-6 weeks later.
- Patients with a larger well-defined abscess: After percutaneous drainage with IV antibiotics is performed, the patient can be discharged with the catheter in place. Interval appendectomy can be performed after the fistula is closed.
- Patients with a multicompartmental abscess: These patients require early surgical drainage.

Although many controversies exist over the nonoperative management of acute appendicitis, antibiotics have an important role in the treatment of patients with this condition. Antibiotics considered for patients with appendicitis must offer full aerobic and anaerobic coverage. The duration of the administration is closely related to the stage of appendicitis at the time of the diagnosis, considering either intraoperative findings or postoperative evolution. According to several studies, antibiotic prophylaxis should be administered before every appendectomy. When the patient becomes afebrile and the white blood cell (WBC) count normalizes,

antibiotic treatment may be stopped. Cefotetan and cefoxitin seem to be the best choices of antibiotics. (See Medications).

Emergency Department Care

The emergency department (ED) clinician must evaluate the larger group of patients who present to the ED with abdominal pain of all etiologies with the goal of approaching 100% sensitivity for the diagnosis in a time-, cost-, and consultation-efficient manner.

Establish IV access and administer aggressive crystalloid therapy to patients with clinical signs of dehydration or septicemia. Patients with suspected appendicitis should not receive anything by mouth. لاشئ بالفم

Administer parenteral analgesic and antiemetic as needed for patient comfort.

The administration of analgesics to patients with acute undifferentiated abdominal pain has historically been discouraged and criticized because of concerns that they render the physical findings less reliable.

However, at least 8 randomized controlled studies have demonstrated that administering opioid analgesic medications to adult and pediatric patients with acute undifferentiated abdominal pain is safe; no study has shown that analgesics adversely affect the accuracy of physical examination. هذا الكلام صحيح لكن للأسف لا يصلح مع المريض المصري .. لأن المريض المصري لو شعر أن الألم قل أو اختفي فسيتقاعس عن العلاج وعن عمل الأشعات والتحاليل وعن العودة للطبيب أو المستشفي ..فيجب أن نفهم هذه النقطة جيدا

Consider ectopic pregnancy in women of childbearing age, and obtain a qualitative beta–human chorionic gonadotropin (beta-hCG) measurement in all cases.

Administer intravenous antibiotics to those with signs of septicemia and to those who are to proceed to laparotomy.

Nonsurgical Treatment

Nonsurgical treatment may be useful when appendectomy is not accessible or when it is temporarily a high-risk procedure. Anecdotal reports describe the success of IV antibiotics in treating acute appendicitis in patients without access to surgical intervention (eg, submariners, individuals on ships at sea).

In a prospective study of 20 patients with ultrasonography-proven appendicitis, symptoms resolved in 95% of patients receiving antibiotics alone, but 37% of these patients had recurrent appendicitis within 14 months.

Preoperative Antibiotics

Preoperative antibiotics have demonstrated efficacy in decreasing postoperative wound infection rates in numerous prospective controlled studies, and they should be administered in conjunction with the surgical consultant. Broad-spectrum gram-negative and anaerobic coverage is indicated (see Medications).

Penicillin-allergic patients should avoid beta-lactamase type antibiotics and cephalosporins. Carbapenems are a good option in these patients.

Pregnant patients should receive pregnancy category A or B antibiotics.

Urgent Versus Emergent Appendectomy

A retrospective study suggested that the risk of appendiceal rupture is minimal in patients with less than 24-36 hours of untreated symptoms, and another retrospective study suggested that appendectomy within 12-24 hours of presentation is not associated with an increase in hospital length of stay, operative time, advanced stages of appendicitis, or complications compared with appendectomy performed within 12 hours of presentation.

Additional studies are needed to demonstrate whether initiation of antibiotic therapy followed by urgent appendectomy is as effective as emergent appendectomy for patients with unperforated appendicitis.

Laparoscopic Appendectomy

Initially performed in 1987, laparoscopic appendectomy has been performed in thousands of patients and is successful in 90-94% of attempts. It has also been demonstrated that laparoscopic appendectomy is successful in approximately 90% of cases of perforated appendicitis. However, this procedure is contraindicated in patients with significant intra-abdominal adhesions.

The 2010 SAGES guideline lists the following conditions as suitable for laparoscopic appendectomy:

- Uncomplicated appendicitis
- Appendicitis in pediatric patients
- Suspected appendicitis in pregnant women

According to the SAGES guideline, laparoscopic appendectomy may be the preferred approach in the following cases:

- Perforated appendicitis
- Appendicitis in elderly patients
- Appendicitis in obese patients

The SAGES guideline states that the laparoscopic approach should be preferred in women of childbearing age with presumed appendicitis.

Diagnostic laparoscopy may be useful in selected cases (eg, infants, elderly patients, female patients) to confirm the diagnosis of appendicitis. This procedure has been suggested for pregnant patients in the first trimester with suspected appendicitis.

If findings are positive, such procedures should be followed by definitive surgical treatment at the time of laparoscopy. Although negative appendectomy does not appear to adversely affect maternal or fetal health, diagnostic delay with perforation does increase fetal and maternal morbidity. Therefore, aggressive evaluation of the appendix is warranted in this group.

Advantages of laparoscopic appendectomy include increased cosmetic satisfaction and a decrease in the postoperative wound-infection rate. Some studies show that laparoscopic appendectomy shortens the hospital stay and convalescent period compared with open appendectomy.

Disadvantages of laparoscopic appendectomy are increased cost and an operating time approximately 20 minutes longer than that of an open appendectomy; however, the latter may resolve with increasing experience with laparoscopic technique. The SAGES guideline recommends practicing a consistent operative method to reduce cost, operating time, and complications.

Complications

Complications of appendicitis may include wound infection, dehiscence, bowel obstruction, abdominal/pelvic abscess, and, rarely, death. Stump appendicitis also occurs rarely; however, at least 36 reported cases of appendicitis in the surgical stump after previous appendectomy exist.

Medication Summary

The goals of therapy are to eradicate the infection and to prevent complications. Thus, antibiotics have an important role in the treatment of appendicitis, and all such. Agents under consideration must offer full aerobic and anaerobic coverage. The duration of the administration is closely related to the stage of appendicitis at the time of the diagnosis.

Antibiotic agents are effective in decreasing the rate of postoperative wound infection and in improving outcome in patients with appendiceal abscess or septicemia. The Surgical Infection Society recommends starting prophylactic antibiotics before surgery, using appropriate spectrum agents for less than 24 hours for nonperforated appendicitis and for less than 5 days for perforated appendicitis. Regimens are of approximately equal efficacy, so consideration should be given to features such as medication allergy, pregnancy category (if applicable), toxicity, and cost.

Penicillins

The penicillins are bactericidal antibiotics that work against sensitive organisms at adequate concentrations and inhibit the biosynthesis of cell wall mucopeptide.

Ampicillin and sulbactam (Unasyn)

This agent is a drug combination of beta-lactamase inhibitor with ampicillin. It is used as a single agent and interferes with bacterial cell wall synthesis during active replication, causing bactericidal activity against susceptible organisms. Ampicillin/sulbactam also has activity against some gram-positive organisms, gram-negative organisms (nonpseudomonal species), and anaerobic bacteria.

Cephalosporins

Cephalosporins are structurally and pharmacologically related to penicillins. They inhibit bacterial cell wall synthesis, resulting in bactericidal activity.

Cefoxitin

This drug is also a second-generation cephalosporin that is indicated as single agent for the management of infections caused by susceptible gram-positive cocci and gram-negative rods. It has a half-life of 0.8 hours.

Cefepime

Cefepime is a fourth-generation cephalosporin. It has gram-negative coverage comparable to ceftazidime but has better gram-positive coverage. Cefepime is a zwitter ion that rapidly penetrates gram-negative cells.

Aminoglycosides

Aminoglycosides have concentration-dependent bactericidal activity. These agents work by binding to the 30S ribosome, inhibiting bacterial protein synthesis.

Gentamicin (Gentacidin, Garamycin)

Gentamicin is an aminoglycoside antibiotic used for gram-negative coverage, as well as in combination with an agent against gram-positive organisms and another one against anaerobes. Gentamicin is not the drug of choice, but consider using this drug if penicillins or other less toxic drugs are contraindicated, when it is clinically indicated, and in mixed infections caused by susceptible staphylococci and gram-negative organisms. This agent may be administered intravenously or ntramuscularly and has numerous regimens; the dose must be adjusted for creatinine clearance and changes in volume of distribution.

Meropenem (Merrem)

Meropenem is a bactericidal broad-spectrum carbapenem antibiotic that inhibits cell wall synthesis. It is used as a single agent and is effective against most gram-positive and gram-negative bacteria.

Fluoroquinolones

These agents can be used to relieve acute undifferentiated abdominal pain in patients presenting to the ED.

Ciprofloxacin (Cipro)

Ciprofloxacin is a fluoroquinolone that inhibits bacterial DNA synthesis and, consequently, growth, by inhibiting DNA gyrase and topoisomerase, which are required for replication, transcription, and translation of genetic material. Quinolones have broad activity against gram-positive and gram-negative aerobic organisms.

Levofloxacin (Levaquin)

Levofloxacin is used for infections caused by various gram-negative organisms, antipseudomonal infections due to multidrug resistant gram-negative organisms.

Anti-infective Agents

Anti-infectives such as metronidazole and tigecycline are effective against many types of bacteria that have become resistant to other antibiotics.

Metronidazole (Flagyl)

Metronidazole has broad gram-negative and anaerobic coverage and is used in combination with aminoglycosides (eg, gentamicin). This agent appears to be absorbed into cells; intermediate metabolized compounds bind DNA and inhibit protein synthesis, causing cell death.

Analgesics

These agents can be used to relieve acute undifferentiated abdominal pain in patients presenting to the ED.

Morphine sulfate

Morphine sulfate is the drug of choice for analgesia because of its reliable and predictable effects, safety profile, and ease of reversibility with naloxone. Various intravenous doses are used; morphine sulfate is commonly titrated to the desired effect.

Carbon Monoxide Toxicity in Emergency Medicine

Background

Carbon monoxide (CO) is a colorless, odorless gas produced by incomplete combustion of carbonaceous material. Commonly overlooked or misdiagnosed, CO intoxication often presents a significant challenge, as treatment protocols, especially for hyperbaric oxygen therapy, remain controversial because of a paucity of definitive clinical studies.

CO is formed as a by-product of burning organic compounds. Although most fatalities result from fires, stoves, portable heaters, and automobile exhaust cause approximately one third of deaths. These often are associated with malfunctioning or obstructed exhaust systems and suicide attempts. Cigarette smoke is a significant source of CO. Natural gas contains no CO, but improperly vented gas water heaters, kerosene space heaters, charcoal grills, hibachis, and Sterno stoves all emit CO. Other sources of CO exposure include propane-fueled forklifts, gas-powered concrete saws, inhaling spray paint, indoor tractor pulls, and swimming behind a motorboat.

Children riding in the back of enclosed pickup trucks seem to be at particularly high risk. Industrial workers at pulp mills, steel foundries, and plants producing formaldehyde or coke are at risk for exposure, as are personnel at fire scenes and individuals working indoors with combustion engines or combustible gases.

Pathophysiology

CO toxicity causes impaired oxygen delivery and utilization at the cellular level. CO affects several different sites within the body but has its most profound impact on the organs (eg, brain, heart) with the highest oxygen requirement.

Toxicity primarily results from cellular hypoxia caused by impedance of oxygen delivery. CO reversibly binds hemoglobin, resulting in relative functional anemia. Because it binds hemoglobin 230-270 times more avidly than oxygen, even small concentrations can result in significant levels of carboxyhemoglobin (HbCO).

CO binds to cardiac myoglobin with an even greater affinity than to hemoglobin; the resulting myocardial depression and hypotension exacerbates the tissue hypoxia. Decrease in oxygen delivery is insufficient, however, to explain the extent of the CO toxicity. Clinical status often does not correlate well with HbCO level, leading some to postulate an additional impairment of cellular respiration.

HbCO levels often do not reflect the clinical picture, yet symptoms typically begin with headaches at levels around 10%. Levels of 50-70% may result in seizure, coma, and fatality.

CO is eliminated through the lungs. Half-life of CO at room air temperature is 3-4 hours. One hundred percent oxygen reduces the half-life to 30-90 minutes; hyperbaric oxygen at 2.5 atm with 100% oxygen reduces it to 15-23 minutes.

History

Misdiagnosis commonly occurs because of the vagueness and broad spectrum of complaints; symptoms often are attributed to a viral illness.

Any of the following should alert suspicion in the winter months, especially in relation to the previously named sources and when more than one patient in a group or household presents with similar complaints.

Symptoms may not correlate well with HbCO levels. For nonfatal nonintentional non –fire-related exposures, the most common symptom was headache (37%) followed by dizziness (18%) and nausea (17%).

- **Acute poisoning**
  - Malaise, flulike symptoms, fatigue - Dyspnea on exertion - Chest pain, palpitations
  - Lethargy - Confusion - Depression - Distractibility
  - Hallucination, confabulation - Agitation
  - Nausea, vomiting, diarrhea - Abdominal pain - Headache, drowsiness
  - Dizziness, weakness, confusion - Visual disturbance, syncope, seizure
  - Fecal and urinary incontinence - Memory and gait disturbances
  - Bizarre neurologic symptoms, coma
- **Chronic exposures** also present with the above symptoms; however, they may present with loss of dentition, gradual-onset neuropsychiatric symptoms, or, simply, recent impairment of cognitive ability.

Physical

Physical examination is of limited value. Inhalation injury or burns should always alert the clinician to the possibility of CO exposure.

- **Vital signs**
  - Tachycardia - Hypertension or hypotension - Hyperthermia

- Marked tachypnea (rare; severe intoxication often associated with mild or no tachypnea)
- **Skin**: Classic cherry red skin is rare (ie, "When you're cherry red, you're dead"); pallor is present more often.
- **Ophthalmologic**
  - Flame-shaped retinal hemorrhages
  - Bright red retinal veins (a sensitive early sign)
  - Papilledema
  - Homonymous hemianopsia
- **Noncardiogenic pulmonary edema**
- **Neurologic and/or neuropsychiatric**
  - Patients display memory disturbance (most common), including retrograde and anterograde amnesia with amnestic confabulatory states.
  - Patients may experience emotional lability, impaired judgment, and decreased cognitive ability.
  - Other signs include stupor, coma, gait disturbance, movement disorders, and rigidity.
  - Patients display brisk reflexes, apraxia, agnosia, tic disorders, hearing and vestibular dysfunction, blindness, and psychosis.
  - Long-term exposures or severe acute exposures frequently result in long-term neuropsychiatric sequelae. Additionally
  - After recovery from the initial incident, patients present several days to weeks later with neuropsychiatric symptoms such as those just described. Two thirds of patients eventually recover completely.
  - MRI changes may remain long after clinical recovery. Predicting and preventing long-term complications and delayed encephalopathy have been the object of recent studies, many of which focus on the role of hyperbaric oxygen therapy.

Causes

- Most unintentional fatalities occur in stationary vehicles from preventable causes such as malfunctioning exhaust systems, inadequately ventilated passenger compartments, operation in an enclosed space, and utilization of auxiliary fuel-burning heaters inside a car or camper.
- Most unintentional automobile-related CO deaths in garages have occurred despite open garage doors or windows, demonstrating the inadequacy of passive ventilation in such situations.
- In the setting of structure fires, CO presents greater risk to firefighters and victims than thermal injury or oxygen deprivation.
- **Most developing countries utilize unvented cookstoves, burning wood, charcoal, animal dung, or agricultural waste. Studies have shown a concurrent rise in HbCO with these types of exposure in developing countries.**

Differential Diagnoses

- Acute Respiratory Distress Syndrome
- Altitude Illness - Cerebral Syndromes
- Depression and Suicide
- Diabetic Ketoacidosis
- Encephalitis
- Gastroenteritis
- Headache, Cluster
- Headache, Tension

- Hypothyroidism and Myxedema Coma - Labyrinthitis - Lactic Acidosis
- Meningitis - Methemoglobinemia - Migraine Headache
- Pediatrics, Headache - Pediatrics, Hypoglycemia - Toxicity, Alcohols
- Toxicity, Narcotics

Laboratory Studies

- HbCO analysis requires direct spectrophotometric measurement in specific blood gas analyzers. Bedside pulse CO-oximetry is now available but requires a special unit and is not a component of routine pulse oximetry.

Prehospital Care .. مثل أي علاج لأي تسمم

Avoid further exposure – Decontamination - Enhance Elimination

- Promptly remove from continued exposure and immediately institute oxygen therapy with a nonrebreather mask.
- Perform intubation for the comatose patient or, if necessary, for airway protection, and provide 100% oxygen therapy.
- Institute cardiac monitoring. Pulse oximetry, although not useful in detecting HbCO, is still important because a low saturation causes an even greater apprehension in this setting.
- Give notification for comatose or unstable patients because rapid or direct transfer to a hyperbaric center may be indicated.
- If possible, obtain ambient CO measurements from fire department or utility company personnel, when present.
- Early blood samples may provide much more accurate correlation between HbCO and clinical status; however, do not delay oxygen administration to acquire them.
- Obtain an estimate of exposure time, if possible.
- Avoid exertion to limit tissue oxygen demand.

Emergency Department Care

- Cardiac monitor: Sudden death has occurred in patients with severe arteriosclerotic disease at HbCO levels of only 20%.
- Pulse oximetry: HbCO absorbs light almost identically to that of oxyhemoglobin. Although a linear drop in oxyhemoglobin occurs as HbCO level rises, pulse oximetry will not reflect it. Pulse oximetry gap, the difference between the saturation as measured by pulse oximetry and one measured directly, is equal to the HbCO level. However, new pulse CO-oximetry units are available which can screen for CO toxicity at the bedside.
- **Continue 100% oxygen therapy until the patient is asymptomatic** and HbCO levels are below 10%. In patients with cardiovascular or pulmonary compromise, lower thresholds of 2% have been suggested.
- Calculate a gross estimate of the necessary duration of therapy using the initial level and half-life of 30-90 minutes at 100% oxygen. Complicated issues of treatment of fetomaternal poisoning are discussed in Special Concerns.

- In uncomplicated intoxications, venous HbCO levels and oxygen therapy are likely sufficient. Evaluate patients with significant cardiovascular disease and initial HbCO levels above 15% for myocardial ischemia and infarction.
- Consider immediate transfer of patients with levels above 40% or cardiovascular or neurologic impairment to a hyperbaric facility, if feasible. Persistent impairment after 4 hours of normobaric oxygen therapy necessitates transfer to a hyperbaric center. Pregnant patients with lower carboxyhemoglobin levels (above 15%) should be considered for hyperbaric treatment.

- Serial neurologic examinations, including funduscopy, CT scans, and, possibly, MRI, are important in detecting the development of cerebral edema. Cerebral edema requires intracranial pressure (ICP) and invasive blood pressure monitoring to further guide therapy. Head elevation, mannitol, and moderate hyperventilation to 28-30 mm Hg PCO_2 are indicated in the initial absence of ICP monitoring. Glucocorticoids have not been proven efficacious, yet the negative aspects of their use in severe cases are limited.
  - Do not aggressively treat acidosis with a pH above 7.15 because it results in a rightward shift in the oxyhemoglobin dissociation curve, increasing tissue oxygen availability. Acidosis generally improves with oxygen therapy.
  - In patients who fail to improve clinically, consider other toxic inhalants or thermal inhalation injury. Be aware that the nitrites used in cyanide kits cause methemoglobinemia, shifting the dissociation curve leftward and further inhibiting oxygen delivery at the tissue level. Combined intoxications of cyanide and CO may be treated with sodium thiosulfate 12.5 g intravenously to prevent the leftward shift.
  - Admit patients to a monitored setting and evaluate acid-base status if HbCO levels are 30-40% or above 25% with associated symptoms.

Consultations

- Hyperbaric oxygen therapy
  - Locate the nearest hyperbaric oxygen center
  - Hyperbaric oxygen therapy (HBO) currently rests at the center of controversy surrounding management of CO poisoning. Increased elimination of HbCO clearly occurs. Certain studies proclaim major reductions in delayed neurologic sequelae, cerebral edema, pathologic central nervous system (CNS) changes, and reduced cytochrome oxidase impairment. Despite these individual claims, systematic reviews have not revealed a clear benefit of HBO, so no clear guidelines for its use have been determined.
  - HBO at 3 atm raises the amount of oxygen dissolved in the serum to 6.8%, enough to sustain cerebral metabolism. Elimination half-life is reduced to 15-23 minutes. Elimination half-life of CO from methylene chloride intoxication of 13 hours at room air temperature is reduced to 5.8 hours. Chambers are small monoplace hulls, allowing space for a single patient in a supine position who can be viewed through a window at the head, or they are acrylic walled and allow full visualization. Many of these monoplace chambers allow for care of critically ill patients, including intravenous lines, arterial lines, and ventilator. Others are large multiplace chambers that permit ventilation equipment and allow medical teams to accompany the patient. A monoplace chamber is shown below.



Monoplace hyperbaric chamber. Courtesy JG Benitez, MD, MPH.

- Treatment regimens usually involve 100% oxygen at 2.4-3 atm for 90-120 minutes. Re-treatment, although controversial, may be performed for acutely and chronically persistent symptoms. One study suggests that degree of acidosis can predict the need for re-treatment.
- Complications of therapy include decompression sickness, sinus and middle ear barotrauma, seizure, progression of pneumothorax to tension pneumothorax, gas embolism, reversible visual refractive changes, and complications related to transport of unstable patients.
- For treatment of complications from therapy, decongestants are useful, prophylactic myringotomy is common and a requirement for intubated patients, and chest tube placement is mandatory with pneumothorax. Exercise caution in patients who have experienced chest compressions, central venous catheterization, intubation, and positive pressure ventilation. Seizures are most often secondary to oxygen toxicity and do not mandate anticonvulsant therapy or discontinuation of HBO therapy.

Febrile convulsion

Background

Febrile seizures are the most common type of seizures observed in the pediatric age group.

The National Institutes of Health described a febrile seizure as, "An event in infancy or childhood usually occurring between three months and five years of age, associated with fever, but without evidence of intracranial infection or defined cause. **حقيقة هنا اختلفت الكتب كثيرا في المرحلة العمرية**" It does not exclude children with prior neurological impairment and neither provides specific temperature criteria nor defines a "seizure."

Pathophysiology

Febrile seizures occur in young children at a time in their development when the seizure threshold is low. This is a time when young children are susceptible to frequent childhood infections such as upper respiratory infection, otitis media, viral syndrome, and they respond with comparably higher temperatures.

Febrile seizures are divided into 2 types: **simple febrile seizures** (which are generalized, last < 15 min and do not recur within 24 h) and **complex febrile seizures** (which are prolonged, recur more than once in 24 h, or are focal). Complex febrile seizures may indicate a more serious disease process, such as meningitis, abscess, or encephalitis.

Viral illnesses are the predominant cause of febrile seizures.

Febrile seizures tend to occur in families. In a child with febrile seizure, the risk of febrile seizure is 10% for the sibling and almost 50% for the sibling if a parent has febrile seizures as well. Although clear evidence exists for a genetic basis of febrile seizures, the mode of inheritance is unclear.

History

- The type of seizure (generalized or focal) and its duration should be described to help differentiate between simple and complex febrile seizures.
- Focus on the history of fever, duration of fever, and potential exposures to illness.
- A history of the cause of fever (eg, viral illnesses, gastroenteritis) should be elucidated.
- Recent antibiotic use is particularly important because partially treated meningitismust be considered.
- A history of seizures, neurologic problems, developmental delay, or other potential causes of seizure (eg, trauma, ingestion) should be sought.

Physical

- The underlying cause for the fever should be sought.
- A careful physical examination often reveals otitis media, pharyngitis, or a viral exanthem.
- Serial evaluations of the patient's neurologic status are essential.
- Check for meningeal signs as well as for signs of trauma or toxic ingestion.

Causes

- Risk factors for developing febrile seizures
  - Family history of febrile seizures - High temperature - Parental report of developmental delay
  - Maternal alcohol intake and smoking during pregnancy has a 2-fold increased risk.
  - Interestingly, no data support the theory that a rapid rise in temperature is a cause of febrile seizures.
- About one third of all children with a first febrile seizure experience recurrent seizures.
  - Risk factors for recurrent febrile seizures include the following:
    - Young age at time of first febrile seizure
    - Relatively low fever at time of first seizure
    - Family history of a febrile seizure in a first-degree relative
    - Brief duration between fever onset and initial seizure
    - Multiple initial febrile seizures during same episode
  - Patients with all 4 risk factors have greater than 70% chance of recurrence. Patients with no risk factors have less than a 20% chance of recurrence.

Differential Diagnoses

- Epidural and Subdural Infections - Epidural Hematoma - Meningitis - Pediatrics, Bacteremia and Sepsis
- Pediatrics, Fever - Pediatrics, Meningitis and Encephalitis - Pediatrics, Status Epilepticus

Prehospital Care *ABC أهم حاجة*

- Patients with active seizures should be treated with airway management, high-flow oxygen, supportive care, and anticonvulsants as necessary.

- Acute treatment such as **rectal diazepam (0.5 mg/kg)** and buccal 0.4-0.5 mg/kg) or intranasal (0.2 mg/kg) are effective and can be given at home for a seizure lasting longer than 5 minutes.
- Patients who are postictal should receive supportive care and antipyretics as appropriate.

Emergency Department Care

- Patients presenting with status epilepticus should be treated with airway management and anticonvulsants as necessary.
- Patients presenting with history and physical examination findings consistent with a simple febrile seizure should have frequent neurologic examinations to monitor mental status.
- Other causes of seizure should be ruled out.
- The cause of the febrile illness should be sought and treated.
- Antipyretics should be considered. Acetaminophen (Tylenol) and ibuprofen (Motrin) are often used.
- Parental anxiety and fear that their child may die or will develop brain damage needs to be addressed with reassurance and education.

Medication Summary

Patients presenting in status epilepticus can be treated with routine seizure medications, including benzodiazepines, phenytoin, and phenobarbital.

Antipyretics

Antipyretics should be used in patients who appear uncomfortable secondary to fever. Antipyretics do not appear to prevent recurrence of febrile seizures.

Acetaminophen

Reduces fever by acting directly on hypothalamic heat-regulating centers, which increases dissipation of body heat via vasodilation and sweating.

Ibuprofen

One of the few NSAIDs indicated for reduction of fever. Inhibits the formation of prostaglandins.

Anticonvulsant agents

Prophylactic treatment with an anticonvulsant agent may be considered for subsequent fever episodes.

Diazepam

Can decrease number of subsequent febrile seizures when given with each febrile episode. Modulates postsynaptic effects of GABA-A transmission, resulting in an increase in presynaptic inhibition. Appears to act on part of the limbic system, the thalamus, and hypothalamus, to induce a calming effect. Also has been found to be an effective adjunct for the relief of skeletal muscle spasm caused by upper motor neuron disorders.

Rapidly distributes to other body fat stores. Twenty minutes after initial IV infusion, serum concentration drops to 20% of C_{max}.

Individualize dosage and increase cautiously to avoid adverse effects. Available as IV, PO, and PR dosage forms.

Lorazepam (Ativan)

Sedative hypnotic with short onset of effects and relatively long half-life.

By increasing the action of gamma-aminobutyric acid (GABA), which is a major inhibitory neurotransmitter in the brain, may depress all levels of CNS, including limbic and reticular formation.

Important to monitor patient's blood pressure after administering dose. Adjust as necessary.

TIA

Background

A transient ischemic attack (TIA) is an acute episode of temporary neurologic dysfunction resulting from focal cerebral ischemia not associated with permanent cerebral infarction.

The clinical symptoms of TIA typically last less than an hour, but prolonged episodes can occur. While the classical definition of TIA included symptoms lasting as long as 24 hours, advances in neuroimaging have suggested that many such cases represent minor strokes with resolved symptoms rather than true TIAs.

A group of cerebrovascular experts proposed a shift from the arbitrary time-based definition of TIA to a tissue-based definition in 2002 with a new definition for TIA as "a brief episode of neurological dysfunction caused by focal brain or retinal ischemia, with clinical symptoms typically lasting less than one hour, and without evidence of acute infarction." This proposed new definition was well received and endorsed by many clinicians. The American Heart Association and American Stroke Association (AHA/ASA) 2009 Guidelines endorsed this new definition, modifying it with the omission of the phrase "typically less than one hour," as there is no time cutoff that reliably distinguishes whether a symptomatic ischemic event will result in tissue infarction.

The AHA/ASA-endorsed definition of TIA is as follows: Transient ischemic attack (TIA) is a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction.

Ruling out metabolic or drug-induced etiologies for symptoms consistent with a TIA is important.

Most importantly, a fingerstick blood glucose should be checked for **hypoglycemia**. Serum electrolytes should be sent to investigate for electrolyte derangements. The following tests are considered emergent: serum chemistry profile, including creatinine; coagulation studies; and complete blood count.

Initial assessment is aimed at excluding emergent conditions that can mimic a TIA, such as **hypoglycemia, seizure, or intracranial hemorrhage**. Laboratory studies, including CBC, coagulation studies, and electrolyte levels, should be obtained. Antithrombotic therapy should be initiated as soon as intracranial hemorrhage has been ruled out

Pathophysiology

The transient ischemic attack (TIA) is characterized by a temporary reduction or cessation of cerebral blood flow in a specific neurovascular distribution that is due to low flow through a partially occluded vessel, an acute thromboembolic event, or stenosis of a small penetrating vessel.

Etiology

The transient ischemic attack (TIA) workup is focused on emergent/urgent risk stratification and management.

Numerous potential underlying causes can be readily identified, including the following:

- Atherosclerosis of carotid and vertebral arteries
- Embolic sources - Valvular disease, ventricular thrombus, and thrombus formation due to atrial fibrillation
- Arterial dissection
- Arteritis - Inflammation of the arteries occurring primarily in elderly persons, especially women; noninfectious necrotizing vasculitis (primary cause); drugs; irradiation; local trauma
- Sympathomimetic drugs (eg, cocaine)
- Mass lesions (eg, tumors, subdural hematomas) - Less frequently cause transient symptoms and more often result in progressive persistent symptoms

Causes of transient ischemic attack in children

TIA etiologies in children, which can differ from those in adults, include the following:

- Congenital heart disease with cerebral thromboembolism (most common)
- Drug abuse (eg, cocaine) - Clotting disorders - Central nervous system infection
- Neurofibromatosis - Vasculitis
- Idiopathic progressive arteriopathy of childhood (moyamoya)
- Fibromuscular dysplasia - Marfan disease - Tuberous sclerosis - Tumor

Prognosis

Patients with TIAs have an increased risk of stroke and death from coronary artery disease (depending on risk factors in the study group, approximately 6-10% per year).

The probability that a person will have a stroke in the 5 years following a TIA is reported to be 24-29%.

Patient Education

It is essential that patients who are being discharged from the hospital first receive clear instruction to ensure understanding of the need for a complete and rapid workup through close follow-up care. The patient who has had a TIA needs to **be educated about lifestyle modification and cardiovascular risk factors**.

Also essential for patients is education on stroke symptoms, the need to call emergency services immediately if any of these symptoms occur, and the contact number for emergency services (911 in the United States).

Additionally, despite program efforts in public education, many patients still do not seek medical attention after experiencing TIA symptoms. A recent population-based study of patients suffering TIA or minor stroke found that 31% of all patients who experienced a recurrent stroke within 90 days of their first TIA or minor stroke did not seek medical attention after the initial event.Public health professionals and physicians need to do more, such as promoting and participating in medical screening fairs and public outreach programs.

History

A transient ischemic attack (TIA) may last only minutes, and symptoms often resolve before the patient presents to a clinician. Thus, historical questions should be addressed not just to the patient but also to family members, witnesses, and emergency medical services (EMS) personnel. Witnesses often perceive abnormalities that the patient cannot, such as changes in behavior, speech, gait, memory, and movement.

Significant medical history questions to elicit any risk factors for relevant underlying disease include questions about the following:

- Recent surgery (eg, carotid, cardiac) - Previous strokes - Seizures - CNS infections
- Use of illicit drugs - Complete medication regimen - Known coagulopathy
- History of arteritis - Noninfectious necrotizing vasculitis, drugs, irradiationThromboembolic risk factors such as carotid artery stenosis, venous or arterial thromboembolism, patent foramen ovale, atrial fibrillation, prior myocardial infarction, or left ventricular dysfunction
- Other known cardiovascular disease

Carefully investigate onset, duration, fluctuation, and intensity of symptoms. Reviewing the patient's medical record is extremely important for identifying deficits from previous strokes, seizures, or cardiac events. The primary care physician can be a reliable resource for insights into previous episodes and workup.

If a patient has a history of associated trauma or cardiac symptoms, the differential diagnosis widens. Pertinent negative items (eg, lack of headache, lack of chest pain, lack of eye pain) in the review of systems also are important.

Carotid or vertebral dissection can occur in association with both major and minor trauma. The patient may provide a history of blunt or torsion injury to the neck. Controversy exists regarding whether chiropractic manipulation or massage therapy increases the risk of arterial dissection.

Physical Examination

The goal of the physical examination is to carefully uncover any neurologic deficits, evaluate for underlying cardiovascular risk factors, and seek any potential thrombotic or embolic source of the event.

A stroke scale prompts the examiner to be thorough and allows different examiners to reliably repeat the examination during subsequent phases of the evaluation. Any neurologic abnormalities should suggest the diagnosis of stroke (or ongoing neurologic event) rather than TIA.

Identify signs of other active comorbidities, including infections (eg, sinusitis, mastoiditis, meningitis) and vasculidities. Carotid arteries can be examined for pulse upstroke, bruit, and the presence of carotid endarterectomy scars.

Funduscopy can identify retinal plaques, retinal pigmentation, and optic disc margins. Pupil reaction to direct and consensual light exposure can be assessed.

In addition to performing **standard auscultation**, examine the chest for the presence of surgical scars, for the presence of a pacemaker/automatic implantable cardioverter defibrillator (AICD), or for other clues that the patient may have a cardiac disorder and increased risk of a cardioembolic phenomenon.

Cardioembolic events are significant causes of TIAs. Identify the rhythm for irregularity or other unusual rhythms and rates, murmurs, or rubs that might suggest valvular disease, atrioseptal defects, or ventricular aneurysm (a source of mural thrombi).

The following signs may be present with cranial nerve dysfunction:

- Ocular dysmotility - Forehead wrinkling asymmetry - Incomplete eyelid closure
- Asymmetrical mouth retraction - Loss of the nasolabial crease - Swallowing difficulty
- Lateral tongue movement - Weak shoulder shrugging - Visual field deficits

The cerebellar system can be tested by assessing ocular movement, gait, and finger-to-nose and heel-to-knee movements, looking for signs of past-pointing and dystaxia, hypotonia, overshooting, gait dystaxia, and nystagmus.

The speech and language system can be tested to assess for both aphasia and dysarthria.

Neurologic Examination

A neurologic examination is the foundation of the TIA evaluation and should focus in particular on the neurovascular distribution suggested by the patient's symptoms. Subsets of the neurologic examination include the following:

- Cranial nerve testing - Somatic motor strength - Somatic sensory testing
- Speech and language testing - Cerebellar system (be sure to see the patient walk)

Mental Status Assessment

Mental status can be assessed formally (Mini-Mental Status Examination, Quick Confusion Scale) or as part of the patient's overall response to questions and interactions with the examiner.

Somatic Motor Testing

Test muscle stretch reflexes of the biceps, triceps, brachioradialis, patellar, and Achilles. In addition, inspect posture and the presence of tremors.

Test the strength of the shoulder girdle, upper extremities, abdominal muscles, and lower extremities, and test passive movement of major joints to look for spasticity, clonus, and rigidity.

Stroke Scale

The National Institutes of Health Stroke Scale (NIHSS) (see Table 1, below) is used mostly by stroke teams for quantifying neurological impairment. It enables the consultant to rapidly determine the severity and possible location of the stroke.

A patient's score on the NIHSS is strongly associated with outcome, and it can help to identify those patients who are likely to benefit from thrombolytic therapy and those who are at higher risk of developing hemorrhagic complications of thrombolytic use.

This scale is easily used and focuses on the following 6 major areas of the neurologic examination:

- level of consciousness - Visual function - Motor function
- Sensation and neglect - Cerebellar function - Language

The NIHSS is a 42-point scale, with minor strokes usually being considered to have a score less than 5. An NIHSS score greater than 10 correlates with an 80% likelihood of visual flow deficits on angiography. Yet, discretion must be used in assessing the magnitude of the clinical deficit; for instance, if a patient's only deficit is being mute, the NIHSS score will be 3. Additionally, the scale does not measure some deficits associated with posterior circulation strokes (ie, vertigo, ataxia).

Vital Signs

Initial vital signs should include the following:

- Temperature - Blood pressure - Heart rate and rhythm
- Respiratory rate and pattern - Oxygen saturation

The examiner should assess the patient's overall health and appearance, making an assessment of the following:

- Attentiveness - Ability to interact with the examiner - Language and memory skills
- Overall hydration status - Development

Diagnostic Considerations

It has been suggested that transient ischemic attacks (TIAs) are a subset of a larger category termed transient neurological attacks (TNAs).

TNAs are defined as episodes of sudden onset of neurological symptoms that completely resolve within 24 hours and have no clear diagnosis. When a TNA is associated with focal symptoms attributed to an arterial territory of the brain, it is consistent with a stroke or TIA and is termed a focal TNA.

In contrast, a nonfocal TNA is a temporary event of diffuse, nonlocalizing, cerebral symptoms that set in suddenly and resolve quickly. Symptoms of nonfocal TNAs may include altered consciousness, nonrotatory dizziness, positive visual phenomena, paresthesias, bilateral weakness, or generalized feelings of unwell with a clinical suspicion for neurological disease. When symptoms are both focal and nonfocal, the term “mixed TNA” would apply.

Nonfocal TNAs have long been considered to be benign. A recent study showed, however, that patients who had suffered a prior nonfocal TNA were at higher risk of stroke and dementia, especially vascular dementia, compared with patients who did not have a history of prior TNA. Although more research is required, this study challenges the idea that nonfocal TNAs are benign events. Further workup should be considered in patients suffering nonfocal TNAs to elucidate the underlying cause of their transient symptoms and to risk stratify them further.

Differential Diagnoses

- Dissection, Carotid Artery - Meningitis - Meningococcal Meningitis

- Multiple Sclerosis - Stroke, Hemorrhagic - Stroke, Ischemic
- Subarachnoid Hemorrhage - Syncope - Syncope and Related Paroxysmal Spells

Approach Considerations

Ruling out metabolic or drug-induced etiologies for symptoms consistent with a transient ischemic attack (TIA) is important. Most importantly, a fingerstick blood glucose should be performed for hypoglycemia. Serum electrolytes should be measured for electrolyte derangements.

Emergent Tests

- Serum chemistry profile, including creatinine - Coagulation studies - Complete blood cell count

Urgent Tests

The following tests typically are helpful and often can be performed on an urgent basis:

- Erythrocyte sedimentation rate (ESR) - Cardiac enzymes - Lipid profile

Screening Tests for Hypercoagulable States

Screening for hypercoagulable states (particularly in younger patients with no known vascular risk factors) include the following:

- Protein C, protein S, antithrombin III activities
- Activated protein C resistance/Factor V Leiden
- Fibrinogen - D-Dimer - Anticardiolipin antibody - Lupus anticoagulant
- Homocysteine - Prothrombin gene G20210A mutation
- Factor VIII - Von Willebrand factor - Plasminogen activator inhibitor-1
- Endogenous tissue plasminogen activator activity

Brain Imaging

National recommendations for urgent evaluation of the patient with a transient ischemic attack (TIA) include imaging of the brain within 24 hours of symptom onset, preferably MRI with diffusion-weighted imaging (DWI), but if this is not available, then a CT scan should be obtained. The cerebral vasculature should be imaged urgently, preferably at the same time as the brain. Brain imaging can identify an area of ischemia in up to 25% of patients, and TIA mimics may be identified as well. Vessel imaging can identify a stenosis or occlusion that requires early intervention.

Noncontrast cranial CT scanning

The noncontrast cranial CT scan is widely and rapidly available and often serves as the initial imaging evaluation. It can aid in diagnosing the following:

- A new area of ischemia or infarction
- Old areas of ischemia
- Intracranial mass such as tumor
- Intracranial bleeding, such as subdural hematoma or intracerebral hemorrhage

Magnetic resonance imaging

MRI is more sensitive than CT for acute ischemia, infarction, previous intracranial bleeding, and other underlying lesions. MRI is less widely available on an acute basis than CT scan, however. The presence of ischemic lesions on an MRI appears to increase the short-term risk of stroke, highlighting its value in acute risk stratification. In addition, a negative DWI image in concert with low-risk clinical features can mark those at minimal short-term stroke risk. Recent data suggest that patients with DWI abnormalities, despite low ABCD2 scores, are at just as high a risk for stroke as patients with high ABCD2 scores but no DWI abnormalities.

Electroencephalography

Electroencephalography (EEG) may be indicated to evaluate for seizure activity.

Vascular Imaging

Vascular imaging for TIA includes Doppler ultrasound, computed tomographic angiography (CTA), and MRA.

Doppler ultrasonography

Carotid Doppler ultrasonography of the neck can be used to identify patients in need of urgent surgical or endovascular therapy. Transcranial Doppler can be a complementary examination evaluating patency of cerebral vessels and collateral circulation.

Angiography

Computed tomographic angiography (CTA) is of increasing value in identifying occlusive disease in the cerebrovascular circulation. Magnetic resonance angiography (MRA) is another alternative for imaging vessels in both the brain and neck. Conventional angiography can be performed when the other modalities are unavailable or yield discordant results.

Cardiac Imaging and Monitoring

Transthoracic or transesophageal echocardiography (TTE/TEE) can evaluate for a cardioembolic source or for risk factors such as patent foramen ovale.

Twelve-lead electrocardiography should be performed as soon as possible after transient ischemic attack (TIA) and can evaluate for dysrhythmias such as atrial fibrillation.

Cardiac monitoring (inpatient telemetry or Holter monitor) is recommended as "useful" in patients without a clear diagnosis after initial brain imaging and electrocardiography.

Lumbar Puncture

Lumbar puncture (LP) may be indicated if subarachnoid hemorrhage, infectious etiology, or demyelinating disease is to be excluded.

Approach Considerations

Rapid transport is essential to evaluate the patient who may have fleeting or stuttering symptoms. **Fingerstick glucose** can quickly rule out hypoglycemia. Intravenous (IV)

access can be established, although transport should not be delayed for this. Collect all the patient's prescription bottles.

The family or witnesses should be instructed to go to the ED, or contact information for these individuals should be obtained. Some communities may have EMS preferentially transfer patients with high-risk stroke symptoms to centers with specific stroke expertise.

Global CNS depression and airway or cardiac compromise are not typically features of a transient ischemic attack (TIA). In fact, the level of consciousness and neurologic examination are expected to be at the patient's baseline. Initial assessment is aimed at excluding emergent conditions that can mimic a TIA, such as hypoglycemia, seizure, or intracranial hemorrhage.

Laboratory studies, including CBC, coagulation studies, and electrolyte levels, should be obtained. Obtain an electrocardiogram (ECG) and evaluate for symptomatic rhythms or evidence of ischemia. Brain imaging is recommended within 24 hours of symptom onset. While MRI with diffusion-weighted imaging (DWI) is preferred, a noncontrast head CT is a reasonable first choice when MRI is not readily available.

Evaluation Timing, Risk Stratification, and Treatment Protocols

Although controversy exists regarding the need for admission, there is no controversy regarding the need for urgent evaluation, risk stratification, and initiation of stroke prevention therapy.

The availability of local resources determines whether this urgent evaluation should occur as an inpatient, in an ED observation unit, or in rapid follow-up. To determine appropriate disposition, the emergency physician should decide on the necessary workup, then discuss with the neurologist or primary care physician how best to ensure that this occurs promptly. In addition to the rapidity of the risk stratification workup, the emergency physician should consider the potential benefit of decreased time to thrombolysis in hospitalized patients diagnosed with TIA who develop a new stroke in the first 24-48 hours after diagnosis.

One randomized controlled trial of an ED diagnostic protocol found that they could reduce cost, length of stay, and provide appropriate risk stratification by performing this workup in an ED observation unit (with neurology consultation) rather than in an inpatient unit.

Restoration of Vital Signs

Vital signs must be obtained promptly and addressed as indicated. Cardiac monitoring can capture a relevant dysrhythmia. Pulse oximetry can evaluate for hypoxia.

Intravenous access (if not already established by EMS) should be obtained. Obtain a fingerstick glucose level, and treat the patient accordingly.

Management of Hypertension

Patients may be significantly hypertensive. Unless there is specific concern for end-organ damage from a hypertensive emergency, blood pressure should be managed conservatively while ischemic stroke is being ruled out.

Acute Ischemic Stroke

For acute ischemic stroke, the American Heart Association recommends initiating antihypertensive therapy only if blood pressure is higher than 220/120 mm Hg or if mean arterial pressure is greater than 130 mm Hg. Unless there is a concerning comorbid cardiac or other condition requiring blood pressure lowering, allowing the patient's blood pressure to autoregulate at a higher level (during the acute phase) may help maximize cerebral perfusion pressure.

Pharmacologic Therapy

Medical management is aimed at reducing both short- and long-term risk of stroke. Antithrombotic therapy should be initiated as soon as intracranial hemorrhage has been ruled out, given the high short-term risk of stroke following TIA. The AHA/ASA guidelines for the prevention of stroke in patients with stroke or transient ischemic attack, issued in 2006 and updated in 2010, are summarized below.

Noncardioembolic transient ischemic attack

Antiplatelet agents are recommended rather than oral anticoagulation as initial therapy. Aspirin (50-325 mg/d), combination aspirin/extended-release dipyridamole, and clopidogrel are all reasonable first-line options (class I recommendation).

Combination aspirin/extended-release dipyridamole (Aggrenox) may be superior to aspirin alone (class IIa recommendation)and can be started within 7 days of the event.

Clopidogrel may be considered instead of aspirin alone (class IIb recommendation). Aspirin in combination with clopidogrel increases the risk of hemorrhage and is not routinely recommended for patients with TIA (class III recommendation).

Cardioembolic transient ischemic attack

In patients with atrial fibrillation after TIA, long-term anticoagulation with warfarin (goal INR, 2-3) is typically recommended. Aspirin, 325 mg/d, is recommended for those unable to take oral anticoagulants. The 2010 AHA/ASA guidelines on stroke prevention after TIA or stroke state that clopidogrel should not be used in combination with aspirin therapy, as the bleeding risk of clopidogrel plus aspirin is similar to that of warfarin.

The 2010 AHA/ASA guidelines also state that it is reasonable for patients with atrial fibrillation who require temporary interruption of oral anticoagulation, but are at high risk for stroke, to be given low molecular weight heparin subcutaneously as bridging therapy.

In acute MI with left ventricular thrombus, oral anticoagulation with warfarin (goal INR 2-3) is reasonable. Aspirin up to 162 mg/d should be used concurrently for ischemic coronary artery disease.

Either oral anticoagulation with warfarin (goal INR 2-3) or antiplatelet therapy may be considered in dilated cardiomyopathy. In rheumatic mitral valve disease, oral anticoagulation with warfarin (goal INR 2-3) is reasonable. Antiplatelet agents would not normally be added to warfarin unless patients experience recurrent embolism despite a therapeutic INR.

According to the 2010 AHA/ASA guidelines, the benefit of warfarin after stroke or TIA in patients with sinus rhythm and cardiomyopathy characterized by systolic dysfunction has not been established.

In mitral valve prolapse, long-term antiplatelet therapy is reasonable. In mitral annular calcification, antiplatelet therapy can be considered. Those with mitral regurgitation can be considered for warfarin or antiplatelet therapy.

In aortic valve disease, antiplatelet therapy may be considered. For mechanical prosthetic valves, oral anticoagulation with warfarin (goal INR 2.5-3.5) is recommended. For those with TIAs despite therapeutic INR, aspirin, 75-100 mg/d, can be added to the regimen. For bioprosthetic valves, patients with TIA and no other source of thromboembolism can be considered for oral anticoagulation with warfarin (goal INR 2-3).

Large-Artery Atherosclerotic Disease

Intracranial Atherosclerosis

The 2010 AHA/ASA guidelines state the following for patients with stroke or TIA due to 50-99% stenosis of a major intracranial artery:

- Aspirin at 50-325 mg/d, rather than warfarin, is recommended.
- Maintenance of blood pressure of less than 140/90 mm Hg and total cholesterol less than 200 mg/dL is recommended.
- Extracranial/intracranial bypass surgery is not recommended.
- Angioplasty or stent placement is investigational and of unknown utility.

Ipsilateral Carotid Artery Stenosis

Patients with TIA and ipsilateral carotid artery stenosis may be candidates for urgent (< 2 wk) carotid endarterectomy. In certain patients, carotid artery stenting is a reasonable alternative. This can be discussed acutely or rapid follow-up arranged.

Extracranial Vertebral Stenosis

Patients with symptoms attributable to extracranial vertebral stenosis may be candidates for endovascular treatment, and again, this should be arranged expediently if available.

According to the AHA/ASA 2010 guidelines, optimal medical treatment for these patients includes antiplatelet and statin therapies as well as risk factor modification. This is also optimal medical treatment for patients with symptomatic extracranial carotid disease.

Long-term Monitoring

Patients selected for outpatient care should have a clear follow-up plan and stroke prevention initiated as above, including antiplatelet medication and risk-factor modification. Antiplatelet agents typically should be initiated as soon as intracranial bleeding is ruled out. As above, the agent to be used varies with the patient and the specific indication.

The following should be included in any long-term monitoring of TIA patients:

- Antihypertensive control should be optimized for patients with hypertension.
- Lipid control should be initiated, potentially including a statin agent.
- Blood glucose control should be optimized for patients with diabetes.
- A smoking-cessation strategy, which may include medication, should be initiated.
- Heavy drinkers should eliminate or reduce alcohol consumption.
- Overweight patients should be encouraged to lose weight.
- All patients should be encouraged to exercise.

Medication Summary

Medical management is aimed at reducing both short- and long-term risk of stroke. Antithrombotic therapy should be initiated as soon as intracranial hemorrhage has been ruled out, given the high short-term risk of stroke following TIA.

Antiplatelet Agents

These agents inhibit platelet function by blocking cyclooxygenase and subsequent aggregation.

Aspirin

Aspirin blocks prostaglandin synthetase action, which, in turn, inhibits prostaglandin synthesis and prevents formation of platelet-aggregating thromboxane A2.

Aspirin 25 mg/dipyridamole 200 mg (Aggrenox)

This combination may be superior to aspirin alone in preventing cardiovascular events following TIAs. Aspirin irreversibly inhibits formation of cyclooxygenase, thus preventing formation of thromboxane A2, a platelet aggregator and vasoconstrictor. Platelet inhibition lasts for the life of a cell (approximately 10 d). Dipyridamole is a platelet adhesion inhibitor that possibly inhibits RBC uptake of adenosine, itself an inhibitor of platelet reactivity.

In addition, it may inhibit phosphodiesterase activity leading to increased cyclic-3',5'-adenosine monophosphate within platelets and formation of the potent platelet activator thromboxane A2. Each capsule contains 25 mg aspirin and 200 mg dipyridamole, for a total of 50 mg aspirin and 400 mg dipyridamole to be given per day.

Clopidogrel (Plavix)

Clopidogrel selectively inhibits ADP binding to platelet receptor and subsequent ADP-mediated activation of glycoprotein GPIIb/IIIa complex, thereby inhibiting platelet aggregation.

Dipyridamole (Persantine)

Dipyridamole is administered to complement usual warfarin therapy. It inhibits platelet adhesion, which may inhibit adenosine uptake by RBCs. It may increase cyclic-3',5'-AMP within platelets and formation of the potent platelet activator thromboxane A2. In addition, it may reduce the risk of stroke when used as monotherapy instead of aspirin.

Ticlopidine (Ticlid)

Ticlopidine is a second-line antiplatelet therapy for patients who cannot tolerate or do not respond to aspirin therapy. In some circumstances, it can be an alternative to clopidogrel.

Anticoagulant

Warfarin (Coumadin)

Warfarin interferes with hepatic synthesis of vitamin K-dependent coagulation factors. It is used for prophylaxis and treatment of venous thrombosis, pulmonary embolism, and thromboembolic disorders.

Pediatric GastroEnteritis

Background

Though often considered a benign disease, acute gastroenteritis remains a major cause of morbidity and mortality in children around the world, accounting for 1.87 million deaths annually in children younger than 5 years, or roughly 19% of all child deaths.Because the disease severity depends on the degree of fluid loss, accurately assessing dehydration status remains a crucial step in preventing mortality. Luckily, most cases of dehydration in children can be accurately diagnosed by a careful clinical examination and treated with simple, cost-effective measures.

Pathophysiology

Adequate fluid balance in humans depends on the secretion and reabsorption of fluid and electrolytes in the intestinal tract; diarrhea occurs when intestinal fluid output overwhelms the absorptive capacity of the gastrointestinal tract. **The 2 primary mechanisms responsible for acute gastroenteritis are (1) damage to the villous brush border of the intestine, causing malabsorption of intestinal contents and leading to an osmotic diarrhea, and (2) the release of toxins that bind to specific enterocyte receptors and cause the release of chloride ions into the intestinal lumen, leading to secretory diarrhea**.

However, even in severe diarrhea, various sodium-coupled solute co-transport mechanisms remain intact, allowing for the efficient reabsorption of salt and water. By providing a 1:1 proportion of sodium to glucose, classic oral rehydration solution (ORS) takes advantage of a specific sodium-glucose transporter (SGLT-1) to increase the reabsorption of sodium, which leads to the passive reabsorption of water. Rice- and cereal-based ORS may also take advantage of sodium-amino acid transporters to increase reabsorption of fluid and electrolytes.

History

The history and physical examination serve 2 vital functions:

(1) differentiating gastroenteritis from other causes of vomiting and diarrhea in children

(2) estimating the degree of dehydration.

In some cases, the history and physical examination can also aid in determining the type of pathogen responsible for the gastroenteritis, though only rarely will this affect management.

- **Diarrhea**: Duration of diarrhea, the frequency and amount of stools, the time since last episode of diarrhea, and the quality of stools.
  Frequent, watery stools are more consistent with viral gastroenteritis, whereas stools with blood or mucus are indicative of a bacterial pathogen. Similarly, a long duration of diarrhea (>14 d) is more consistent with a parasitic or noninfectious cause of diarrhea.
- **Vomiting**: Duration of vomiting, the amount and quality of vomitus (eg, food contents, blood, bile), and time since the last episode of vomiting. When symptoms of vomiting predominate, one should consider other diseases such as gastroesophageal reflux disease (GERD), diabetic ketoacidosis, pyloric stenosis, acute abdomen, or urinary tract infection.
- **Urination**: Increase or decrease in frequency of urination measured by number of wet diapers, time since last urination, color and concentration of urine, and presence of dysuria. Urine output may be difficult to determine with frequent watery stools.
- **Abdominal pain**: Location, quality, radiation, severity, and timing of pain, per report of parents and/or child. In general, pain that precedes vomiting and diarrhea is more likely to be due to abdominal pathology other than gastroenteritis.
- **Signs of infection**: Presence of fever, chills, myalgias, rash, rhinorrhea, sore throat, cough. These may indicate evidence of systemic infection or sepsis.
- **Appearance and behavior**: Weight loss, quality of feeding, amount and frequency of feeding, level of thirst, level of alertness, increased malaise, lethargy, or irritability, quality of crying, and presence or absence of tears with crying.
- **Antibiotics:** History of recent antibiotic use increases the likelihood of*Clostridium difficile*.
- **Travel**: History of travel to endemic areas may make prompt consideration of organisms that are relatively rare in the United States, such as parasitic diseases or cholera.

Physical

- **General**: Weight, ill appearance, level of alertness, lethargy, irritability as depicted in the video below.
- **Head, ears, eyes, nose, and throat (HEENT):** Presence or absence of tears, dry or moist mucous membranes, and whether the eyes appear
- **Cardiovascular**: Heart rate and quality of pulses
- **Respiratory**: Rate and quality of respirations (The presence of deep, acidotic breathing suggests severe dehydration
- **Abdomen**: Abdominal tenderness, guarding, and rebound, bowel sounds. Abdominal tenderness on examination, with or without guarding, should prompt consideration of diseases other than gastroenteritis.
- **Back**: Flank/costovertebral angle (CVA) tenderness increases the likelihood of pyelonephritis.
- **Rectal**: Quality and color of stool, presence of gross blood or mucus
- **Extremities**: Capillary refill time, warm or cool extremities

- **Skin:** Abdominal rash may indicate typhoid fever (infection with *Salmonella typhi*), whereas jaundice might make viral or toxic hepatitis more likely. The slow return of abdominal skin pinch suggests decreased skin turgor and dehydration , while a doughy feel to the skin may indicate hypernatremia.

Causes يجب معرفة السبب حتي تعالج بطريقة صحيحة

Identifying the specific etiologic agent responsible for the acute gastroenteritis rarely changes management. However, it may be helpful to differentiate between viral, bacterial, parasitic, and noninfectious causes of diarrhea.

By far, **viruses** remain the most common cause of acute gastroenteritis in children, both in the developed and developing world. Rotavirus represents the most important viral pathogen worldwide, responsible for 29% of all diarrhea-related deaths.Rotavirus infection follows seasonal variation, with an increased incidence in winter and decreased incidence in summer.

Viral gastroenteritis typically presents with low-grade fever and vomiting followed by copious watery diarrhea (up to 10-20 bowel movements per day), with symptoms persisting for 3-8 days.

In developed countries, bacterial pathogens account for a small portion, perhaps 2-10%, of all cases of pediatric gastroenteritis.

Relative to viral gastroenteritis, **bacterial disease** is more likely to be associated with high fevers, shaking chills, bloody bowel movements (dysentery), abdominal cramping, and fecal leukocytes.

In developing countries, enterotoxigenic *Escherichia coli* (ETEC) remains the most important bacterial cause of acute gastroenteritis in children, followed by*Campylobacter*, *Salmonella*, and *Shigella* species, while also causing the majority of traveler's diarrhea cases in all age groups.Unlike other bacterial causes of gastroenteritis, ETEC is unlikely to cause dysentery.

Clostridium difficile has emerged as an important cause of antibiotic-associated diarrhea in children. Any antibiotic can trigger infection with *C difficile*, though penicillins, cephalosporins, and clindamycin are the most likely causes.*Since 50% of neonates and young infants are colonized with* C difficile, *symptomatic disease is unlikely in children younger than 12 months.*

Parasites remain yet another source of gastroenteritis in young children, with*Giardia* and *Cryptosporidium* the most common causes in the United States. **Parasitic gastroenteritis** generally present with watery stools but can be differentiated from viral gastroenteritis by a protracted course or history of travel to endemic areas.

Differential Diagnoses

- Diabetic Ketoacidosis
- Gastritis and Peptic Ulcer Disease
- Giardiasis

- Hemolytic Uremic Syndrome - Hepatitis - Inflammatory Bowel Disease - Pancreatitis
- Pediatrics, Appendicitis - Pediatrics, Foreign Body Ingestion - Pediatrics, Intussusception
- Pediatrics, Pyloric Stenosis - Pediatrics, Urinary Tract Infections and Pyelonephritis - Shock, Septic

Laboratory Studies

The vast majority of children presenting with acute gastroenteritis do not require serum or urine tests, as they are unlikely to be helpful in determining the degree of dehydration. In a meta-analysis of 6 studies, only serum bicarbonate (< 17) had statistically significant positive and negative likelihood ratios for detecting moderate dehydration.

Clinically significant electrolyte abnormalities are rare in children with moderate dehydration. However, any child being treated with intravenous fluids for severe dehydration should have baseline electrolytes, bicarbonate, and urea/creatinine drawn. Laboratory tests are also indicated in patients with moderate dehydration whose history and physical examination are inconsistent with straightforward gastroenteritis.

Fecal leukocytes and stool culture may be helpful in children presenting with dysentery. Children older than 12 months of age with a recent history of antibiotic use should have stool tested for *C difficile* toxins. Those with a history of prolonged watery diarrhea (>14 d) or travel to an endemic area should have stool sent for ova and parasites.

Any child with evidence of systemic infection should have a complete workup, including CBC and blood cultures. If indicated, urine cultures, chest radiography, and/or lumbar puncture should be performed.

Imaging Studies

Abdominal films are not indicated in the management of acute gastroenteritis. If the clinician suspects a diagnosis other than acute gastroenteritis based on history and physical examination findings, appropriate imaging modalities should be pursued.

Table 2. Assessment of Dehydration According to the World Health Organization*

Severe Dehydration	Two of the following signs: -Lethargic or unconscious -Sunken eyes -Not able to drink or drinking poorly -Skin pinch goes back very slowly

Some Dehydration	Two of the following signs: -Restless, irritable -Sunken eyes -Thirsty, drinks eagerly -Skin pinch goes back slowly
No Dehydration	Not enough of the above signs to classify as some or severe dehydration

Prehospital Care

Children with acute gastroenteritis rarely require intravenous access. In those presenting with circulatory collapse due to severe dehydration or sepsis, **intravenous access should be obtained and followed by an immediate 20 mL/kg bolus of normal saline.**

Emergency Department Care

The American Academy of Pediatrics, the European Society of Pediatric Gastroenterology and Nutrition (ESPGAN), and the World Health Organization **all recommend oral rehydration solution (ORS) as the treatment of choice for children with mild-to-moderate gastroenteritis in both developed and developing countries**, based on the results of dozens of randomized, controlled trials and several large meta-analyses.

Initial care in the emergency department should focus on correction of dehydration. The type and amount of fluid given should reflect the degree of dehydration in the child.

Minimal or no dehydration

No immediate treatment is required.

If the child is breastfed, the mother should be encouraged to breastfeed more frequently than usual and for longer at each feed.

If the child is not exclusively breastfed, then oral maintenance fluids (including clean water, soup, rice water, yogurt drink, or other culturally appropriate fluid) should be given at a rate of approximately 500 mL/day for children younger than 2 years, 1000 mL/day for children aged 2-10 years, and 2000 mL/day for children older than 10 years.

In addition, ongoing fluid losses should be replaced with 10 mL/kg body weight of additional ORS for each loose stool and 2 mL/kg body weight of additional ORS for each episode of emesis (both for breastfed and nonbreastfed children).

Mild-to-moderate dehydration

Children should be given 50-100 mL/kg of ORS over a 2- to 4-hour period to replace their estimated fluid deficit, with additional ORS given to replace ongoing losses (10

mL/kg body weight for each stool and 2 mL/kg body weight for each episode of emesis).

After the initial rehydration phase, patients may be transitioned to maintenance fluids as described above.

ORS should be given slowly by the caregiver or parent using a teaspoon, syringe, or medicine dropper at a rate of 5 mL every 1-2 minutes. If tolerated by the patient, the rate of ORS delivery can be increased slowly over time.

For patients who do not tolerate ORS by mouth, nasogastric (NG) feeding is a safe and effective alternative.

Multiple clinical trials have found NG rehydration to be as efficacious as intravenous rehydration, but more cost-effective and with fewer adverse events.

Patients should be reassessed frequently by the clinician to ensure adequacy of oral intake and resolution of the various signs and symptoms of dehydration.

Severe dehydration

Severe dehydration constitutes a medical emergency requiring immediate resuscitation with intravenous fluids. Intravenous access should be obtained,

And patients should be administered a **bolus of 20-30 mL/kg lactated Ringer's (LR) or normal saline (NS).**

If pulse, perfusion, and/or mental status do not improve, a second bolus should be administered.

After this, the patient should be given an infusion of 70 mL/kg LR or NS over 5 hours (children < 12 months) or 2.5 hours (older children). If no peripheral veins are available, an intraosseous line should be placed. Serum electrolytes, bicarbonate, urea/creatinine, and glucose levels should be sent.

Once resuscitation is complete and mental status returns to normal, rehydration should continue with ORS as described above, as it has been shown to decrease the rate of hyponatremia and hypernatremia when compared with intravenous rehydration.

Type of ORS

A large *CochraneDatabase of Systematic Reviews* meta-analysis confirmed several earlier studies showing that reduced osmolarity ORS (osmolarity < 250 mmol/L) is associated with fewer treatment failures, lower stool output, and less frequent vomiting compared with standard osmolarity ORS for patients with noncholera gastroenteritis.However, patients with cholera appear to have higher rates of hyponatremia with reduced osmolarity ORS compared with standard osmolarity ORS, without any of the added benefits seen in patients with noncholera gastroenteritis.

In developing countries, clinicians can use WHO ORS sachets or a homemade solution of 3 g (1 tsp) salt and 18 g (6 tsp) sugar added to 1 liter of clean water. ملعقة ملح +6معالق سكر علي لتر ماء نظيف

New research suggests that polymer-based ORS, made from complex carbohydrates such as rice, wheat, or maize, may reduce stool output and length of diarrhea compared with glucose-based ORS. With these solutions, carbohydrates are slowly digested in the small intestine, releasing glucose to facilitate sodium uptake without adding a significant osmotic load to bowel contents

Feeding and nutrition

In general, children with gastroenteritis should be returned to a normal diet as rapidly as possible. Early feeding reduces illness duration and improves nutritional outcome.

Breastfed infants should continue breastfeeding throughout the rehydration and maintenance phases of acute gastroenteritis. Formula fed infants should restart feeding at full strength as soon as the rehydration phase is complete (ideally in 2-4 h). Weaned children should restart their normal fluids and solids as soon as the rehydration phase is complete. **Fatty foods and foods high in simple sugars should be avoided.**

For most infants, clinical trials have found no benefit of lactose-free formulas over lactose-containing formulas. Similarly, highly specific diets, such as the BRAT (bananas, rice, applesauce, and toast) diet, have not been shown to improve outcomes and may provide suboptimal nutrition for the patient.

Medication Summary

The goals of pharmacotherapy are to reduce morbidity, to prevent complications, and for prophylaxis. Antidiarrheal (ie, kaolin-pectin) and antimotility agents (ie, loperamide) are contraindicated in the treatment of acute gastroenteritis in children because of their lack of benefit and increased risk of side effects, including ileus, drowsiness, and nausea.علي الرغم من هذا الكلام لكن الكاولين والبكتن من أكثر الأدوية كتابة في النزلات المعوية وفكرتهم بيخلوا البرز ماسك فبيطمن الأم .

Probiotics are live microbial feeding supplements commonly used in the treatment and prevention of acute diarrhea. <u>Possible mechanisms of action include synthesis of antimicrobial substances, competition with pathogens for nutrients, modification of toxins, and stimulation of nonspecific immune responses to pathogens</u>. Two large systematic reviews have found probiotics (especially Lactobacillus GG) to be effective in reducing the duration of diarrhea in children presenting with acute gastroenteritis. Because probiotic preparations vary widely, estimating the effectiveness of any single preparation is difficult.متاحة في السوق المصري .. ابحث في أطلس الأدوية عنها

One meta-analysis, including 18 published studies, all conducted in developing countries, found **zinc** supplementation to be effective in reducing the duration and severity of diarrhea in children with acute gastroenteritis. **The WHO recommends zinc supplementation (10-20 mg/d for 10-14 d) for all children younger than 5 years with acute gastroenteritis, though little data exist to support this recommendation for children in developed countries.** **أصبح الزنك متاحا الان في السوق المصري ومهم جدا في علاج الحالات ويجب كتابته**

Vaccines

Rotavirus vaccine (RotaTeq, Rotarix)

Clinical trials reported that the vaccines prevented 74-78% of all rotavirus gastroenteritis cases, nearly all severe rotavirus gastroenteritis cases, and nearly all hospitalizations due to rotavirus.

Antimicrobials

Because most cases of acute gastroenteritis in developed and developing countries are due to viruses, antibiotics are generally not indicated.

Even in cases (eg, dysentery) where a bacterial pathogen is suspected, antibiotics may prolong the carrier state (*Salmonella*) or may increase the risk of hemolytic uremic syndrome (enterohemorrhagic *E coli*).

In patients with positive stool assays or high clinical suspicion for *C difficile*, the offending antibiotic should be stopped immediately. **Metronidazole** (30 mg/kg/d divided qid for 7 d) can be used as a first-line agent, with oral vancomycin reserved for resistant infections.

Though generally not recommended for children younger than 8 years, tetracycline (50 mg/kg/d divided qid for 3 d) and doxycycline (6 mg/kg single dose) remain the treatment of choice for cholera. Alternative treatments with good efficacy include erythromycin and ciprofloxacin.

For patients with ova and parasites confirming infection with *Giardia*, metronidazole (35-50 mg/kg/d divided q8h) remains the drug of choice. **Nitazoxanide** oral suspension (aged 1-3 y: 100 mg q12h for 3 d, aged 4-11 y: 200 mg q12h for 3 d) is as effective as metronidazole and has the added benefit of treating other intestinal parasites, such as *Cryptosporidium*.

Metronidazole (Flagyl)

Recommended as the treatment of choice for mild-to-moderate cases of *C difficile*colitis. Provides effective therapy, with reported response rates from 95-100%.

.Metronidazole IV may be administered to those patients who cannot tolerate PO medications because of its potential to accumulate in the inflamed colon. IV route is not as effective as PO.

Nitazoxanide

Inhibits growth of *C parvum* sporozoites and oocysts and *G lamblia* trophozoites. Elicits antiprotozoal activity by interference with the pyruvate: ferredoxin oxidoreductase (PFOR) enzyme-dependent electron transfer reaction, which is essential to anaerobic energy metabolism

Antiemetics

vitamin B6 for Pediatric

Domperidone جرعته في الشراب 1سم لكل كيلو في اليوم وتقسم علي 3 مرات قبل الرضاعة أو قبل الأكل

Metaclopromide خليه آخر حاجة ولازم تحسب الجرعة للطفل وتديله الجرعة بنفسك ولا تكررها خوفا من دخوله في

Extrapyramidal syndrome

Further Inpatient Care

Inpatient admission should be considered for all children with acute gastroenteritis in the following situations:

- Signs of severe dehydration are present.
- Caregivers are unable to manage oral rehydration or provide adequate care at home.
- Substantial difficulties exist in administering ORS, such as intractable vomiting or inadequate ORS intake.
- Failure of treatment occurs, such as worsening diarrhea or dehydration, despite adequate ORS intake.
- Factors are present necessitating closer observation, such as young age, decreased mental status, or uncertainty of diagnosis.
- Children with mild-moderate dehydration, age < 6 months, or high frequency of stools/vomits should be monitored in the emergency department for a minimum of 4-6 hours before discharge.

Further Outpatient Care

- Parents should be instructed to continue providing maintenance ORS fluids at home as needed. Breastfeeding and formula feeding should be continued for infants, and children should be encouraged to return to a regular diet as rapidly as possible.
- Parents should be instructed to look for the various signs of dehydration outlined above, such as change in mental status, decreased urine output, sunken eyes, absence of tears, dry mucous membranes, and slow return of abdominal skin pinch.
- Parents should seek medical attention if dehydration returns, oral intake is inadequate, or if their child develops worsening abdominal pain, fever >101°F, or prolonged diarrhea lasting longer than 14 days.

Helicobacter Pylori

Background

In 1983, Warren (a biologist) and Marshall (a clinician) described *Helicobacter pylori (HP)*. At first, they named the bacterium *Campylobacter pyloridis*. Later, it was named *Campylobacter pylori*. Since then, a large number of reports have been produced on *H pylori* and its pathogenetic potential.

In fact, although peptic ulcer disease is the most studied disease related to *H pylori* infection, this bacterium is seemingly involved in the pathogenesis of several extragastric diseases, such as mucosa-associated lymphoid tissue lymphomas (MALTomas), coronaritis, gastroesophageal reflux disease (GERD), iron deficiency anemia, skin disease, and rheumatologic conditions. However, at present, many of

these associations remain largely uncertain, and the debate to confirm or refute causality related to these associations is still open.

H pylori infection occurs more frequently in developing countries than in industrialized countries. *H pylori* strains differ in their potential to cause diseases. Although anyone can develop a microscopic gastritis, only a minority of infected persons develop ulcers or other diseases.

Pathophysiology

The most common route of *H pylori* infection is either oral-to-oral (stomach contents are transmitted from mouth to mouth) or fecal-to-oral (from stool to mouth) contact. Parents and siblings seem to play a primary role in transmission.

History

In the authors' opinion, no significant differences in the presence and frequency of symptoms, such as nausea, vomiting, pain, heartburn, or diarrhea, occur in patients who are infected with *H pylori* and patients who are not. No definite evidence demonstrates a clear relationship between the symptoms of the *H pylori* -associated gastritis and abdominal pain or dyspeptic symptoms from other conditions. Of patients, 30-35% have no symptoms.

Physical

No specific clinical signs have been described in patients with *H pylori* infection.

- Patients may feel dyspepsia or abdominal discomfort, such as during gastritis or with epigastric pain (eg, duodenal ulcers).
- In some cases, patients may feel hungry in the morning and may have halitosis.

Causes

- *H pylori* infection causes atrophic and even metaplastic changes in the stomach.
- The bacterial adhesion appears to result in tyrosine phosphorylation and is specific for gastric cells.
- The adhesion of *H pylori* to the gastric cells causes a direct decrease in mucosal levels of glutathione, a fundamental molecule in the maintenance of the cellular redox status and in the molecular regulation of host immune responses.
  However, the LPS of *H pylori* may induce the production of autoantibodies that are able to worsen atrophy in the corpus mucosa and cause a concomitant increase in parietal cell antibodies. Such events are accompanied by a decrease in anti-*H pylori* immunoglobulin titers. This process leads to a scenario of severe atrophy without bacterial colonization combined with high levels of autoantibodies against gastric parietal cells.
- A number of reports show the close association between *H pylori* infection and low-grade gastric MALTomas.
- Giannakis and colleagues demonstrated that *H pylori* may adapt to gastric stem cells, influencing their biology and contributing to tumorigenesis of the stomach.

Differential Diagnoses

– Duodenal Ulcers - Gastric Cancer - Gastric Ulcers - Gastrinoma - Gastritis, Acute

- Gastritis, Atrophic - Gastritis, Chronic - Gastritis, Stress-Induced - Gastroesophageal Reflux Disease
- Lymphoma, Non-Hodgkin

Laboratory Studies

- *H pylori* fecal antigen test
  - This novel rapid test is based on monoclonal antibody immunochromatography of stool samples. The test has been reported to be very specific (98%) and sensitive (94%).
  - The results are positive in the initial stages of infection and can be used to detect eradication after treatment.
  - Although the *H pylori* fecal antigen test is an interesting tool, information about the cost of the test is pending.
- Carbon 13 urea breath test
  - The carbon 13 urea breath test (UBT) is based on the detection of the products created when urea is split by the organism.
  - Patients are asked to drink urea (usually with a beverage) labeled with a carbon isotope (carbon 13 or carbon 14). After a certain duration, the concentration of the labeled carbon is measured in the breath. The concentration is high only when urease is present in the stomach. Because the human stomach does not produce urease, such a reaction is possible only with *H pylori* infection.
  - The breath test is expensive but is becoming increasingly more available.
  - Other problems include false-negative results due to infection with coccoid forms of *H pylori* that do not produce as much urease or intake of antibiotics, bismuth, histamine 2 (H2) blockers, or proton pump inhibitors.
- *H pylori* serology
  - The serology test has a high (>90%) specificity and sensitivity. It is currently based on the quantitation of immunoglobulin G antibodies against*H pylori* by the means of an enzyme-linked immunosorbent assay.
  - It is useful for detecting a newly infected patient, but it is not a good test for follow-up of treated patients because the results do not indicate present infection with *H pylori*. The antibody titer may remain elevated for a long time after *H pylori* eradication. The number of false-positive results is age related and increases with age.

Medical Care

Only treat patients who have a test result positive for *H pylori* infection. Carefully educate patients regarding the importance of completing the prescription and about the potential adverse effects of the medication. Importantly, consider possible antibiotic resistance when selecting the treatment regimen.

The US Food and Drug Administration has approved some regimens, which are now accepted internationally, for the treatment of *H pylori* infection in patients with peptic ulcer disease, both gastric and duodenal. These regimens are also known as **triple therapies** and have reported cure rates from 85-90%.

Administer triple therapies for 10-14 days. **The treatment regimens are omeprazole, amoxicillin, and clarithromycin (OAC) for 10 days**;

bismuth subsalicylate, metronidazole, and tetracycline (BMT) for 14 days;

and **lansoprazole, amoxicillin, and clarithromycin (LAC)**, which has been approved for either 10 days or 14 days of treatment.

H pylori eradication rates were higher for a 7-day antibiotic regimen containing lansoprazole, amoxicillin, and clarithromycin (LAC), when used as first-line therapy compared with levofloxacin, amoxicillin, and lansoprazole (LAL).Additionally, LAC did not achieve a higher rate of eradication than LAL as second-line therapy; thus, consideration of the sequence of administering antibiotic regimens for *H pylori* is important.

All the eradication treatments have a high incidence of certain adverse effects (eg, nausea, metallic taste). If skin rash, vomiting, or diarrhea occurs, discontinue treatment.

Surgical Care

Surgery is not required for patients with *H pylori* infection, but it may be considered in patients with severe complications, such as cancer.

Medication Summary

The goals of pharmacotherapy are to eradicate the microorganism, to prevent complications, and to reduce morbidity. Triple therapies are used. Worldwide, accepted treatment regimens are BMT, LAC, and OAC. BMT regimen is based on the administration of bismuth subsalicylate, metronidazole, and tetracycline. Add an H2-receptor antagonist for an additional 4 weeks. LAC regimen is based on the administration of lansoprazole, amoxicillin, and clarithromycin. OAC regimen is based on the administration of omeprazole, amoxicillin, and clarithromycin.

Increasing resistance to antibiotics has made alternative treatments necessary. In a phase 3 trial conducted by Malfertheiner et al, quadruple therapy (omeprazole plus a single 3-in-1 capsule containing bismuth subcitrate potassium, metronidazole, and tetracycline) was tested against standard therapy in adults with recorded *H pylori* infection. This study suggests quadruple therapy provides superior eradication with similar safety and tolerability to standard therapy.

Antidiarrheals

The approved antidiarrheal for this infection is bismuth subsalicylate. It has both antisecretory and antimicrobial activity.

Bismuth subsalicylate (Bismatrol, Pepto-Bismol)

Has cytoprotective effect on GI mucosa, probably due to stimulation of prostaglandin production and modulation of immune response. In addition, has been demonstrated that some deposits (probably bismuth salts) appear on both surfaces of the cell wall of *H pylori* after < 1 h. Such deposits induce distortion and vacuolization of the bacterial cell and loss of adherence of *H pylori* from antral epithelium.

Antibiotics

Use agents known to be effective against *H pylori.*

Metronidazole (Flagyl)

Reduced to its active form intracellularly only by anaerobic organisms, then disrupts helical structure of DNA and inhibits bacterial nucleic acid synthesis.

Clarithromycin (Biaxin)

Inhibits bacterial growth, possibly by blocking dissociation of peptidyl tRNA from ribosomes, causing RNA-dependent protein synthesis to arrest.

Amoxicillin (Amoxil)

Inhibits final stage of bacterial cell wall synthesis due to binding to specific PBPs on inner part of bacterial wall, leading to bacterial lysis.

Proton pump inhibitors

Bind to proton pump of parietal cell, inhibiting secretion of hydrogen ions into gastric lumen. Relieve pain and heal peptic ulcers more rapidly than H2 antagonists.

Lansoprazole

Works by inhibiting the H^+/K^+ -ATPase enzyme system of gastric parietal cells.

Omeprazole

Decreases gastric acid secretion by inhibiting parietal cell H^+/K^+ -ATP pump.

H2 receptor blockers

Reversible competitive blockers of histamine at H2 receptors, particularly those in gastric parietal cells, wherein they inhibit acid secretion. H2 antagonists are highly selective, do not affect the H1 receptors, and are not anticholinergic agents. Proton pump inhibitors are usually preferred.

Ranitidine (Zantac)

Reduces basal and nocturnal gastric acid secretion by competitive inhibition of binding of histamine to receptors (H2 receptor) on gastric parietal cells. Although not effective as single agents for the eradication of *H pylori*, appears to increase systemic absorption of bismuth subsalicylate.

Famotidine

Competitively inhibits histamine at H2 receptor of gastric parietal cells, resulting in reduced gastric acid secretion, gastric volume, and hydrogen ion concentrations.

Gout and pseudogout

Background

Gout and pseudogout are the 2 most common crystal-induced arthropathies. They are debilitating illnesses in which recurrent episodes of pain and joint inflammation are caused by the formation of crystals within the joint space and deposition of crystals in soft tissue. If untreated, these disorders can lead to joint destruction and renal damage. Rarely, gout can produce significant ocular findings.The incidence of age-related macular degeneration (ARMD) is higher in patients with gout.

Gout is inflammation caused by monosodium urate monohydrate (MSU) crystals. Pseudogout is inflammation caused by calcium pyrophosphate (CPP) crystals and is sometimes referred to as calcium pyrophosphate disease (CPPD).

Although gout is associated with hyperuricemia, the level of uric acid does not itself precipitate gout; rather, acute changes in the level of uric acid cause gout. Most individuals with hyperuricemia do not have gout, but if high uric acid levels go untreated, 90% of patients develop gout within 30 years.

Hyperuricemia is found in 90% of individuals with gout, but it is also found in patients taking diuretics and even in those taking low doses of aspirin.

Primary gout is related to underexcretion or overproduction of uric acid. Secondary gout is related to myeloproliferative diseases or their treatment, therapeutic regimens producing hyperuricemia, renal failure, renal tubular disorders, lead poisoning, hyperproliferative skin disorders, enzymatic defects (eg, deficient hypoxanthine-guanine phosphoribosyl transferase, and glycogen storage diseases).

Gout is definitively diagnosed based on the demonstration of urate crystals in aspirated synovial fluid. Classic radiographic findings may also be diagnostic. (See Workup).

Improvements in early diagnosis and the availability of definitive treatment have significantly improved the prognosis of gout, as evidenced by the declining incidence of disabling chronic tophaceous gout. However, tophaceous gout may still develop because of misdiagnosis, poor management, medication intolerances, and/or poor patient compliance.

Treatment of gout is important to relieve pain, prevent disease progression, and prevent tissue deposition of uric acid (eg, in the kidneys) that may produce kidney stones or urate nephropathy.

Treatment of the acute phase of pseudogout is identical to that of gout. Unlike gout, however, no specific therapeutic regimen exists to treat the underlying cause of pseudogout, but colchicine and hydroxychloroquine are effective for prophylaxis.

Historical background مرض الملوك

Gout is one of the oldest diseases in the medical literature. Since the time of the Greeks, many authors have written about gout as the result of personal excess; its association with a diet rich in meat and alcohol gained it the sobriquet, “the king of diseases and the disease of kings”. However, among the abstinent was John Milton, who lived a life of rigorous self-discipline and yet, to his anger and despair, suffered from what commonly was regarded as just punishment of the dissolute.

Pathophysiology

Gout can be considered a disorder of metabolism that allows uric acid/urate to accumulate in blood and tissues. When tissues become supersaturated, the urate salts precipitate, forming crystals. In addition, the crystals also are less soluble under acid conditions, so any condition predisposing to acidosis also precipitates urate crystals.

Urate initially precipitates in the form of needlelike crystals. The light-retarding (phase-shifting) characteristics of urate crystals allow them to be recognized by polarizing microscopy.

Many conditions and drugs have been associated with an increase in plasma (and subsequent synovial) urate levels. A genetic predisposition for the disease exists.

Etiology

Gout develops in the setting of excessive stores of uric acid in the form of monosodium urate. Uric acid is an end-stage by-product of purine metabolism. Humans remove uric acid primarily by renal excretion.

When excretion is insufficient to maintain serum urate levels below the saturation level of 6.8 mg/dL (with some variability depending on temperature and pH), hyperuricemia may develop, and urate can crystallize and deposit in soft tissues.

Ninety percent of patients with gout develop excess urate stores due to an inability to excrete sufficient amounts of normally produced uric acid in the urine (underexcretion). The remaining patients either overconsume purines or produce excessive amounts of uric acid endogenously (overproduction).

Overproduction of uric acid may also occur in disorders that cause high cell turnover with release of purines, which are present in high concentration in cell nuclei. These disorders include myeloproliferative and lymphoproliferative disorders, psoriasis, hemolytic anemias, pernicious anemia, and ineffective erythropoiesis (as in B-12 deficiency). Cell lysis from chemotherapy for malignancies, especially those of the hematopoietic or lymphatic systems, can raise uric acid levels, as can excessive exercise and obesity.

Common causes of secondary gout due to underexcretion of uric acid include renal insufficiency, lead nephropathy (saturnine gout), starvation or dehydration, hypothyroidism, hyperparathyroidism, drugs, and chronic ethanol (especially beer and hard liquor) abuse. These disorders should be identified and corrected, if possible.

Comorbidities, including hypertension, diabetes, renal insufficiency, hypertriglyceridemia, hypercholesterolemia, diabetes, obesity, and early menopause, are associated with a higher incidence of gout.

Foods that are rich in purines include anchovies, sardines, sweetbread, kidney, liver, and meat extracts. Consumption of fructose-rich foods and beverages (eg, those sweetened with high-fructose corn syrup) are associated with an increased risk of gout in both men and women.

Individual gout flares are often triggered by acute increases or decreases in urate levels that may lead to the production, exposure, or shedding of crystals that are not coated with apo B or apo E. This can result from acute alcohol ingestion, acute overindulgence in foods high in purines, rapid weight loss, starvation, trauma, emotional stress, or hemorrhage.

Similarly, flares can be precipitated by additions of or changes in dosage of medications that raise or lower uric acid levels, such as loop or thiazide diuretics, aspirin, allopurinol, or uricosurics.

Medications that increase uric acid levels via effects on renal tubular transport include loop and thiazide diuretics, low-dose aspirin, and cyclosporine A. Agents that lower levels of uric acid include radiocontrast dyes, xanthine oxidase inhibitors (eg, allopurinol, febuxostat), and uricosurics (eg, probenecid, sulfinpyrazone).

Although the pathophysiology, clinical presentation, and acute-phase treatment of gout and pseudogout are very similar, the underlying causes of the 2 diseases are very different. Many cases of pseudogout are idiopathic, but pseudogout has also been associated with aging, trauma, and many different metabolic abnormalities, the most common of which are hyperparathyroidism and hemochromatosis. Pseudogout attacks have been reportedly induced by etidronate disodium therapy and angiography.

Gout and renal disease

Although patients with gout often have other risk factors for renal disease, including hypertension and diabetes, chronic urate nephropathy can contribute to renal insufficiency. Chronic urate nephropathy in patients with chronic tophaceous gout can result from the deposition of urate crystals in the medullary interstitium and pyramids, resulting in an inflammatory reaction that can lead to fibrotic changes. This process is characterized by hyperuricemia that is disproportional to the degree of renal impairment and is associated with a benign urinary sediment.

Prognosis

Gout is associated with considerable morbidity, with acute episodes often causing incapacitation. However, gout that is treated early and properly carries an excellent prognosis if patient compliance is good.

With early treatment, gout should be totally controlled. If attacks recur, successful uric acid adjustment (requiring lifelong use of uricosuric or allopurinol medication) usually suppresses further activity. During the first 6-24 months of uricosuric or allopurinol therapy, acute attacks of gout may occur.

Chronic injury to intra-articular cartilage leaves the joints more susceptible to subsequent joint infections.

Draining tophi can become secondarily infected. Untreated chronic tophaceous gout can lead to severe joint destruction and renal impairment. Deposition of MSU crystals in the kidney can result in inflammation and fibrosis, leading to reduced renal function or chronic nephropathy.

Acute attacks of pseudogout usually resolve within 10 days. Prognosis for resolutions of acute attacks is excellent. Some patients experience progressive joint damage with functional limitation.

Hyperuricemia and gout are associated with an increased overall likelihood of mortality. Whether this is directly attributable to hyperuricemia or gout or to gout-associated diseases (eg, insulin resistance, type 2 diabetes mellitus, abdominal obesity, hypercholesterolemia, hypertension) has been much debated.

Although no evidence has shown that gout or hyperuricemia causes any of these disorders, elevated urate levels have been shown to correlate with blood pressure in

adolescents.Among middle-aged men, hyperuricemia with gout was a significant independent risk for death due to cardiovascular disease.

Patient Education

Patients with severe hyperuricemia should avoid food with high purine content. Moderation in food and alcohol is advised. Early recognition of acute attacks is critical, as intervention with medication is much more effective earlier in the attack.

History

The spontaneous onset of pain, edema, and inflammation in the metatarsal-phalangeal joint of the great toe (podagra) is highly suggestive of acute crystal-induced arthritis. Podagra is the initial joint manifestation in 50% of gout cases. Eventually, it is involved in 90% of cases.



Gout. Acute podagra due to gout in an elderly man.

Podagra is not synonymous with gout, however. Podagra may be observed in patients with pseudogout, sarcoidosis, gonococcal arthritis, psoriatic arthritis, and reactive arthritis.

Other than the great toe, the most common sites of gouty arthritis are the ankle, wrist, and knee. In early gout, only 1 or 2 joints are usually involved. Consider the diagnosis in any patient with acute monarticular arthritis of any peripheral joint except the glenohumeral joint of the shoulder, in which a crystal-induced arthritis is more likely to be due to pseudogout.

In addition to the shoulder, the most common sites of pseudogout arthritis are the knee and wrist. Case reports have documented carpal tunnel syndrome as an initial presentation of pseudogout.

Case reports of pseudogout forming masses in the spinal ligamentum flavum have been documented.These have led to both single and multi-level myelopathy.

Although crystal-induced arthritis is most commonly monarticular, polyarticular acute flares are not rare, and many different joints may be involved simultaneously or in rapid succession. Multiple joints in the same limb often are involved, as when inflammation begins in the great toe and then progresses to involve the midfoot and ankle.

Gout attacks begin abruptly and reach maximum intensity within 8-12 hours. The joints are red, hot, and exquisitely tender; even a bed sheet on the swollen joint is uncomfortable. The onset of symptoms in pseudogout is usually more insidious and may occur over several days.

Untreated, the first attacks resolve spontaneously in less than 2 weeks. A history of intermittent inflammatory arthritis, in which the joints return to normal between attacks,

is typically caused by crystalline disorders and is characteristic of gouty arthritis early in its course.

Gout initially presents as polyarticular arthritis in 10% of patients. Elderly women, particularly women with renal insufficiency who are taking a thiazide diuretic, can develop polyarticular arthritis as the first manifestation of gout. These attacks may occur in coexisting Heberden and Bouchard nodes. Such patients may also develop tophi more quickly, occasionally without prior episodes of acute gouty arthritis.

The pattern of symptoms in untreated gout changes over time. The attacks become more polyarticular. Although more joints may become involved, inflammation in a given joint may become less intense. More proximal and upper-extremity joints become involved. Attacks occur more frequently and last longer.

Eventually, patients may develop chronic polyarticular arthritis, sometimes nearly symmetrical, that can resemble rheumatoid arthritis. Indeed, chronic polyarticular arthritis that began as an intermittent arthritis should prompt consideration of a crystalline disorder in the differential diagnoses.

Acute flares of gout can result from situations that lead to increased levels of serum uric acid, such as the consumption of beer or liquor, overconsumption of foods with high purine content, trauma, hemorrhage, dehydration, or the use of medications that elevate levels of uric acid. Acute flares of gout also can result from situations that lead to decreased levels of serum uric acid, such as the use of radiocontrast dye or medications that lower the levels of uric acid, including allopurinol and uricosurics.

Patients with gout are profoundly more likely to develop renal stones than are healthy individuals (by a factor of 1000); therefore, they may have a history of renal colic and of hematuria. Indeed, renal stones may precede the onset of gout in 40% of affected patients. While 80% of these patients may have stones composed entirely of uric acid, 20% may develop calcium oxalate or calcium phosphate stones with a uric acid core.

Because gout is frequently present in patients with the metabolic syndrome (eg, insulin resistance or diabetes, hypertension, hypertriglyceridemia, and low levels of high-density lipoproteins) and because the presence of these associated disorders can lead to coronary artery disease, these problems should be sought and treated in patients diagnosed with gout.

Fever, chills, and malaise do not distinguish cellulitis or septic arthritis from crystal-induced arthritis because all 3 illnesses can produce these signs and symptoms. A careful history may uncover risk factors for cellulitis or septic arthritis, such as possible exposure to gonorrhea, a recent puncture wound over the joint, or systemic signs of disseminated infection.

Physical Examination

Patients with an acute attack of gout or pseudogout most often present with involvement of a single joint. However, examine all joints to determine if the patient's arthritis is monoarticular or polyarticular. Involved joints have all the signs of inflammation: swelling, warmth, erythema, and tenderness.

The erythema over the joint may resemble cellulitis; the skin may desquamate as the attack subsides. The joint capsule becomes quickly swollen, resulting in a loss of range of motion of the involved joint.

During an acute gout attack, patients may be febrile, particularly if it is an attack of polyarticular gout.

However, look for sites of infection that may have seeded the joint and caused an infectious arthritis that can resemble or coexist with acute gouty arthritis.

Migratory polyarthritis is a rare presentation. Polyarticular gout commonly involves the small joints of the fingers and toes, as well as the knees. An inflammatory synovial effusion may be present. Uncommonly, acute gout may present as carpal tunnel syndrome.

Chronic arthritis with tenderness and swelling, with or without redness, warmth, or joint damage, may be present.

Posterior interosseous nerve syndrome has been reported because of elbow inflammation causing compression of the nerve. In patients presenting with a swollen elbow and inability to extend the fingers actively, this should be considered. Treatment with intra-articular steroids has led to resolution of the nerve palsy in a case report.

Tophi

Although gout typically causes joint inflammation, it can also cause inflammation in other synovial-based structures such as bursae and tendons. Tophi are collections of urate crystals in the soft tissues. They tend to develop after about a decade in untreated patients who develop chronic gouty arthritis. Tophi tend to develop earlier in women, particularly those receiving diuretics.

The classic location of tophi is along the helix of the ear but they can be found in multiple locations, including the fingers, toes, prepatellar bursa, and along the olecranon, where they can resemble rheumatoid nodules. Rarely, a creamy discharge may be present.The finding of a rheumatoid nodule in a patient with a negative rheumatoid factor result or a history of drainage from a nodule should prompt consideration of gout in the differential diagnoses.

Complications

Complications of gout include the following:

- Severe degenerative arthritis - Secondary infections
- Urate or uric acid nephropathy - Nerve or spinal cord impingement - Increased susceptibility to infection
- Urate or uric acid nephropathy - Renal stones
- Nerve or spinal cord impingement - Fractures

Diagnostic Considerations

The history and physical examination alone cannot reliably determine the cause of new-onset acute monoarticular arthritis. Septic arthritis, gout, and pseudogout can present in very similar ways.

Certain clinical presentations are so characteristic of gout that attempts have been made to accurately diagnose or exclude gout without joint aspiration. Janssens et adeveloped a diagnostic rule for this purpose, which included the following:

- Male sex - Previous arthritis attack
- Onset within 1 day - Joint redness
- First metatarsophalangeal joint involvement
- Hypertension or one or more cardiovascular diseases
- A serum uric acid level of more than 5.88 mg/dL

Differential Diagnoses

- Arthritis as a Manifestation of Systemic Disease - Bursitis
- Cellulitis - Chondrocalcinosis - Hyperparathyroidism
- Nephrolithiasis - Paronychia - Rheumatoid Arthritis
- Septic Arthritis - Tenosynovitis

Serum Uric Acid

Measurement of serum uric acid is the most misused test in the diagnosis of gout. The presence of hyperuricemia in the absence of symptoms is not diagnostic of gout. In addition, as many as 10% of patients with symptoms due to gout may have normal serum uric acid levels at the time of their attack. Thus, the correct diagnosis of gout can be missed if the joint is not aspirated. Remember that situations that decrease uric acid levels can trigger attacks of gout. In such cases, the patient's prior medical records may reveal prior elevations of uric acid.

Urinary Uric Acid

A 24-hour urinary uric acid evaluation is generally performed if uricosuric therapy is being considered. If patients excrete more than 800 mg of uric acid in 24 hours while eating a regular diet, they are overexcretors and thus overproducers of uric acid. These patients (approximately 10% of patients with gout) require allopurinol instead of probenecid to reduce uric acid levels.

Patients who excrete more than 1100 mg in 24 hours should undergo close renal function monitoring because of the risk of stones and urate nephropathy.

In patients in whom probenecid is contraindicated (eg, those with a history of renal stones or renal insufficiency), a 24-hour urine test of uric acid excretion does not need to be performed because the patient clearly will need allopurinol.

Blood Studies

Obtaining an accurate measure of the patient's renal function before deciding on therapy for gout is important, since the serum creatinine evaluation alone can underestimate renal dysfunction in elderly patients or in patients with low muscle mass.

Glucose measurement is useful because patients with gout are at an increased risk of developing diabetes mellitus. Liver function studies are important because abnormal results may affect the selection of therapy.

The WBC count may be elevated in patients during the acute gouty attack, particularly if it is polyarticular.

Hypertriglyceridemia and low high-density lipoproteins are associated with gout.

Pseudogout attacks can be triggered by many metabolic abnormalities.

Thus, patients who have an initial attack of arthritis with CPP crystals should have a workup including a chemistry screen; magnesium, calcium, and iron levels; and thyroid function tests.

Radiography

Plain radiographs may show findings consistent with gout, but these findings are not diagnostic. Early in the disease, radiographs are often normal or show only soft-tissue swelling. Radiographic findings characteristic of gout, which generally do not appear within the first year of disease onset, consist of punched-out erosions or lytic areas with overhanging edges, as shown in the image below.



Gout. Radiograph of erosions with overhanging edges.

Erosions with overhanging edges generally are considered pathognomonic for gout but also can be found in amyloidosis, multicentric reticulohistiocytosis, and type IIA hyperlipoproteinemia.

Haziness suggestive of tophi can be seen in late gout, and tophi may calcify. Characteristics of erosions that are typical of gout but not of rheumatoid arthritis include the following:

- Maintenance of the joint space
- Absence of periarticular osteopenia
- Location outside the joint capsule



Gout. Plain radiograph showing chronic tophaceous gouty arthritis in the hands.

Approach Considerations

Gout is managed in 3 stages: (1) treating the acute attack, (2) providing prophylaxis to prevent acute flares, and (3) lowering excess stores of urate to prevent flares of gouty arthritis and to prevent tissue deposition of urate crystals.

Treatment of Acute Attacks

The temptation to treat patients without a proven diagnosis must be resisted, as septic arthritis may clinically resemble gout or pseudogout.

Unrecognized septic arthritis can lead to loss of life or of limb. Distinguishing septic arthritis from crystal-induced arthritis is not possible without an examination of joint fluid.

Acute treatment of proven crystal-induced arthritis is directed at **relief of the pain and inflammation**.

Nonsteroidal anti-inflammatory drugs (NSAIDs), colchicine, and ACTH are the mainstays of treatment. The choice is based primarily on any concomitant health problems (eg, renal insufficiency, peptic ulcer disease). Colchicine, a classic treatment, is now rarely indicated.

Patients should be instructed to go on a diet if obese, to stop drinking beer, and to avoid purine-rich foods.

Therapy to control the underlying hyperuricemia generally is contraindicated until the acute attack is controlled (unless kidneys are at risk because of unusual uric acid load). Further, control of hyperuricemia generally is not pursued for a single attack

Starting therapy to control hyperuricemia during an acute attack may intensify and prolong the attack. If the patient has been on a consistent dose of probenecid or allopurinol for more than 2 weeks at the time of the acute attack, the drug should be continued at that dose during the attack.

For urate crystals within the ocular tissue, treatment is directed at reducing both hyperuricemia and ocular inflammation.

Nonsteroidal anti-inflammatory drugs

NSAIDs are the drugs of choice in most patients with acute gout who do not have underlying health problems. Although indomethacin is the traditional NSAID of choice for acute gout (unless the patient is elderly, because of the potential for adverse CNS effects in this age group), most NSAIDs can be used. Select an agent with a quick onset of action, but do not use aspirin because it can alter uric acid levels and potentially prolong and intensify an acute attack. Cyclooxygenase-2 (COX-2) inhibitors have been used with success.

Avoid NSAIDs in patients who have a history of peptic ulcer disease or GI bleeding, patients with renal insufficiency, patients with abnormal hepatic function, patients taking warfarin (selective COX-2 inhibitors can be used), and patients in the intensive

care unit who are predisposed to gastritis. Use NSAIDs cautiously in patients with diabetes and those who are receiving concomitant angiotensin-converting enzyme (ACE) inhibitors.

NSAIDs are prescribed at full dosage for 2-5 days to control the acute attack, and the dose is reduced to approximately one half to one fourth of that amount once the acute attack is controlled. Taper the dose down over approximately 2 weeks.

Gout symptoms should be absent for at least 2 days before the NSAID is discontinued. Anti-inflammatory therapy is continued (at low doses) for 6-18 months in patients starting medication to lower uric acid levels, as this treatment precipitates gout in up to 50% of patients.

Patient compliance of only 20% indicates that repeated reinforcement of the treatment regimen is necessary.

Colchicine

Although colchicine was once the treatment of choice for acute gout, it is now a second-line approach because of its narrow therapeutic window and risk of toxicity. Colchicine therapy must be initiated within 24 hours of onset of the acute attack to be effective. When used in classic hourly dosing regimens in acute gout, colchicine causes adverse GI effects, particularly diarrhea and vomiting, in 80% of patients. This dosing regimen has been superseded (see below).

Dosing recommendations for colchicine in acute gout therapy have been modified in recent years because of an increased awareness of its toxicities. The most recent recommendations have been trending toward lowered daily and cumulative doses.

Colchicine should not be used if the glomerular filtration rate (GFR) is less than 10 mL/min, and the dose should be decreased by at least half if the GFR is less than 50 mL/min. Colchicine should also be avoided in patients with hepatic dysfunction, biliary obstruction, or an inability to tolerate diarrhea.

A clinical response to colchicine is not pathognomonic for gout and may occur in patients with pseudogout, sarcoid arthropathy, psoriatic arthritis, or calcific tendonitis.

Prophylaxis

The standard dose of colchicines for prophylaxis is 0.6 mg bid, but lower doses have also been suggested. In patients with renal insufficiency, this dose may need to be decreased to daily or every-other-day administration. Even in prophylactic doses, colchicines can cause marrow toxicity and neuromyopathy in the setting of renal insufficiency.

Long-term use of colchicine can lead to a muscle weakness associated with elevated levels of creatine kinase due to a drug-induced neuromyopathy, particularly in patients with renal insufficiency.

Prophylaxis with colchicine can be started during an acute attack. Lowering uric acid with either allopurinol or probenecid can precipitate attacks of gout. When used prophylactically, colchicine can reduce such flares by 85%. Patients with gout may be

able to abort an attack by taking a single colchicine tablet at the first twinge of an attack.

Corticosteroids

Corticosteroids can be given to patients with gout who cannot use NSAIDs or colchicine, but adrenocorticotropic hormone (ACTH) would be preferred. Steroids can be given orally, intravenously, intramuscularly, intra-articularly, or indirectly via ACTH.

ACTH at 40 IU IM can be given to induce corticosteroid production by the patient's own adrenal glands. Such a regimen does not depend on the patient to properly taper prednisone.

Prednisone can be given at a dose of approximately 40 mg for 1-3 days and then tapered over approximately 2 weeks. Tapering more rapidly can result in a rebound flare. Prophylaxis for steroid adverse effects (eg, osteoporosis) is recommended.

Using parenteral corticosteroids confers no advantage unless the patient cannot take oral medications.

Intra-articular, long-acting (depot) corticosteroids are particularly useful in patients with a monoarticular flare to help reduce the systemic effects of oral steroids. Ensuring that the joint is not infected prior to injecting intra-articular corticosteroids is particularly important.

Treatment of Chronic Gout

In many cases, patients who have a first attack of gout should undergo therapy with agents that lower uric acid, given the high risk for further inflammatory attacks and the potential for destructive tophaceous deposition in the bone, synovium, and kidney, even without episodes of acute inflammation. However, some rheumatologists advocate waiting for the second attack to initiate therapy to lower uric acid levels because not all patients experience a second attack and because some patients may need to be convinced they need life-long therapy.

The risk of a second attack of gout after the first attack is 62% after 1 year, 78% after 2 years, and 93% after 10 years. The decision to begin therapy depends partly on the baseline serum uric acid levels (>9 mg/dL denotes a higher risk for recurrent gouty arthritis and tophi).

Long-term management of gout is focused on lowering uric acid levels. The goal of therapy is to lower serum uric acid levels to approximately 6 mg/dL or less. A prospective cohort study of 211 patients with gout found that maintenance of serum uric acid levels below 6 mg/dl resulted in disappearance of urate crystals in synovial fluid, resorption of tophi and cessation of acute attacks. Maintenance for 5 years is apparently sufficient in the absence of tophi to permit complete dissolution of all urate crystals. Use of diuretics is to be avoided, as they reduced ability to lower uric acid levels below 7 mg/dl in 17% of study participants.

Avoiding the use of medications that elevate uric acid in patients with gout is prudent.

Thus, in patients with hypertension, other agents are preferable to a thiazide diuretic. The angiotensin receptor blocker losartan should be considered. Losartan (is uricosuric at 50 mg/d. However, medications that elevate uric acid can still be used, if required, by making appropriate adjustments of allopurinol or probenecid doses.

If the patient has tophaceous disease, probenecid should not be used. Urinary alkalization and the recommendation to ingest copious amounts of fluid are adjunctive.

As both probenecid and allopurinol change serum and tissue uric acid levels, they may predispose patients to acute gouty attacks. Colchicine or low-dose NSAID treatment is used for 6-24 months to reduce this undesired effect. In patients who cannot take colchicine or NSAIDs, low doses of prednisone can be considered.

Monotherapy with colchicine may help prevent flares of inflammatory arthritis but does not prevent the accumulation of uric acid in the joints, which can lead to further joint destruction.

Agents that lower uric acid levels should not be initiated during an acute attack, because this may lead to a more intense and prolonged attack. Typically, they should be started a few weeks after the attack has resolved and with the protection of prophylactic colchicine to prevent another attack.

If the patient develops a gout flare after beginning therapy with a uric acid–lowering agent, the agent should not be discontinued because this will only cause another flux in the uric acid level, which may prolong and intensify the attack.

Some rheumatologists prefer probenecid whenever possible because it has fewer significant adverse effects than allopurinol. Probenecid can be used in most middle-aged patients with gout who are otherwise healthy**. Patients who use probenecid need to drink 2 L of fluid daily at the inception of therapy in order to reduce their urinary concentration and thereby reduce the risk of renal stones**

Allopurinol

Allopurinol blocks xanthine oxidase and thus reduces the generation of uric acid. Therefore, it should be used in patients who overproduce uric acid and in patients at risk of tumor lysis syndrome to prevent renal toxicity during therapy for malignancies. It is the most effective urate-lowering agent. However, alcohol can interfere with the effectiveness of allopurinol.

Approximately 3-10% of patients taking allopurinol develop dyspepsia, headache, diarrhea, and/or pruritic maculopapular rash. Less frequently, patients taking allopurinol can develop allopurinol hypersensitivity, which carries a mortality rate of 20-30%.

Allopurinol is also associated with the drug rash with eosinophilia and systemic symptoms (DRESS) syndrome. DRESS syndrome affects the liver, kidney, and skin. It is a delayed-hypersensitivity response occurring 6-8 weeks after beginning allopurinol.

The underlying mechanism is thought to be a cell-mediated immunity to allopurinol and its metabolites. Although occurrence is 0.4 %, the rate of organ failure and death is high. Treatment is with intravenous *N*- acetyl cysteine and steroids.

Allopurinol should be discontinued in patients who develop a rash. In patients with a history of drug eruptions due to allopurinol, both oral and intravenous desensitization regimens can be considered.

In most patients, start at 100 mg per day (50 mg in patients with renal insufficiency) and adjust the dose monthly according to the uric acid level until the goal of a uric acid level of 6 mg/dL or less is achieved.

Febuxostat

Febuxostat, a nonpurine selective inhibitor of xanthine oxidase, is a potential alternative to allopurinol in patients with gout. Febuxostat is administered orally and is metabolized mainly in the liver. In contrast, allopurinol and its metabolites are excreted primarily by the kidney. Therefore, febuxostat can be administered in patients with renal insufficiency with no dosage adjustment. Its efficacy and side-effect profile otherwise appears similar to that of allopurinol.

Uricase

Nonrecombinant urate-oxidase (uricase) is used in Europe to prevent severe hyperuricemia induced by chemotherapy in patients with malignancies, as well as in selected patients with treatment-refractory gout. Short-term use of such agents in patients with severe tophaceous could debulk the total-body urate load, allowing for maintenance with probenecid or allopurinol.

Other therapeutic options

Patients with allopurinol hypersensitivity can often tolerate oxypurinol, which is a metabolite of allopurinol. It is available on a "compassionate use" basis. Cross-reactivity with allopurinol can occur.

Benzbromarone is an effective uricosuric agent that may eventually become available. However, it can cause fulminant hepatotoxicity.

The angiotensin receptor blocker losartan and the triglyceride-lowering agent micronized fenofibrate have moderately potent uricosuric effects. They should therefore be considered in patients with gout who also require treatment for hypertension and hypertriglyceridemia.

Vitamin C, with its uricosuric effect, may reduce the serum concentration of uric acid. In one study, 500 mg/day for 2 months reduced uric acid by a mean of 0.5 mg/dL. Vitamin C treatment should be avoided with nephrolithiasis or urate nephropathy, cystinuria, or penicillamine.

Diet and Activity

As uric acid is a breakdown product of purine, high-purine foods should be avoided, or consumed only in moderation. Foods very high in purines include hearts, sweetbreads (eg, pancreas, thymus), smelt, sardines, and mussels. Foods moderately high in

purines include anchovies, trout, haddock, scallops, mutton, veal, liver, bacon, salmon, kidneys, and turkey.

Purines are found in all protein foods. All sources of purines cannot and should not be eliminated, but a low-protein diet may be helpful.

Overall, purine restriction reduces serum uric acid levels by only 1 mg/ml, at significant psychological impact. Diet modifications are rarely able to lower uric acid levels sufficiently to prevent further attacks and accumulation of urate.

Patients with gout should avoid beer and hard liquor because they elevate levels of uric acid and therefore can precipitate attacks of gout. Indeed, heavy drinkers are much more likely to have recurrent gout attacks, even with allopurinol therapy. Moderate wine intake is not associated with an increased gout flares.

Increasing dairy intake, folic acid intake, and coffee consumption may reduce gout flares.

Particularly because of the association of gout with atherosclerosis, the diagnosis of gout may be a good time to advise a low-cholesterol, low-fat diet if otherwise appropriate for the patient. While such a diet may help uric acid levels, such advice should be given primarily to help prevent atherosclerosis.

Weight reduction in patients who are obese can improve hyperuricemia. Ketosis-inducing diets (eg, fasting) should be avoided, however.

Guidelines for the management of gout have been issued by the American College of Rheumatology (ACR) regarding the following

Urate-lowering therapy

- Chronic tophaceous gouty arthropathy (CTGA)
- Analgesic and anti-inflammatory management of acute gouty arthritis
- Drug prophylaxis of acute attacks
- Guidance on the use of febuxostat and pegloticase

Primary recommendation:

- More intensive education of patients on diet, lifestyle choices, treatment objectives, and management of comorbidities such as obesity, excessive alcohol intake, urolithiasis, chronic kidney disease, uric acid overproduction, and lead intoxication.

First-line pharmacologic urate-lowering therapy:

- ACR guidelines recommend treating patients with a xanthine oxidase inhibitor, such as allopurinol, as the first-line pharmacologic urate-lowering therapy.
- Recommended goal is to reduce serum urate to < 6 mg/dL; initial allopurinol dosage should be no greater than 100 mg/day. This should be followed by a gradual increase of the maintenance dose, which can safely exceed 300 mg even in patients with chronic kidney disease.

Prescreening:

- To avoid allopurinol toxicity, the guidelines recommend considering HLA-B*5801 prescreening of patients at particularly high risk for severe adverse reaction to allopurinol.

CTGA:

- The ACR guidelines recommend combination therapy, with 1 xanthine oxidase inhibitor (allopurinol or febuxostat) and 1 uricosuric agent, when target urate levels are not achieved.
- Use probenecid as an alternative first-line urate-lowering drug in the setting of contraindications or intolerance to at least 1 xanthine oxidase inhibitor (except in patients with creatinine clearance < 50 mL/min).
- Use pegloticase in patients with severe gout disease who do not respond to standard urate-lowering therapy.

Therapy and prophylactic anti-inflammatory treatment for acute gouty arthritis:

- Initiate pharmacologic therapy within 24 hours of onset of an acute gouty arthritis attack while continuing urate-lowering therapy without interruption.
- Nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, or oral colchicine is the recommended first-line treatment for acute gout; combinations of these medications can be used for severe or unresponsive cases.
- To prevent acute gout flares that may accompany the early stages of urate-lowering therapy, the ACR guidelines recommend oral colchicine or low-dose NSAIDs, as long as there is no medical contraindication or lack of tolerance.

Long-Term Monitoring

After diagnosis and treatment of an acute gouty arthritis episode, the patient should return for a follow-up visit in approximately 1 month to be evaluated for therapy to lower serum uric acid levels.

If uric acid–lowering therapy is begun, patients should be seen every 1-2 months while adjusting the dose of medications to achieve the target uric acid level of 5-6 mg/dL. Once this level is achieved and maintained, patients can be seen every 6-12 months.

Crying Babies

There's no getting around it: Babies cry. It's how they communicate hunger, pain, fear, a need for sleep, and more.

So how are parents supposed to know what their baby is trying to tell them? It can be tricky to interpret your child’s cries, especially at first.

Here are the most common reasons babies cry. If your little one is wailing and you don't know why, work your way down the list. Chances are you'll find something that helps.

1. Hunger

This is probably the first thing you think of when your baby cries.

Learning to recognize the signs of hunger will help you start your baby's feedings before the crying stage. Some signs to watch for in newborns: fussing, smacking of lips, rooting (a newborn reflex that causes babies to turn their head toward your hand when you stroke their cheek), and putting their hands to their mouth.

2. A dirty diaper

Some babies let you know right away when they need to be changed. Others can tolerate a dirty diaper for quite a while.

3. Needs sleep

Aren't babies lucky? When they're tired they can simply go to sleep – anytime, anywhere. Or so adults like to think.

In reality, it's harder for them than you might think. Instead of nodding off, babies may fuss and cry, especially if they're overly tired.

4. Wants to be held

Babies need a lot of cuddling. They like to see their parents' faces, hear their voices, and listen to their heartbeats, and can even detect their unique smell. Crying can be their way of asking to be held close.

You may wonder if you'll spoil your baby by holding him so much, but during the first few months of life that isn't possible. To give your arms some relief, try wearing your baby in a front carrier or sling.

5. Tummy troubles (gas, colic, and more)

Tummy troubles associated with gas or colic can lead to lots of crying. In fact, the rather mysterious condition called colic is defined as inconsolable crying for at least three hours a day, at least three days a week, at least three weeks in a row.

If your baby often fusses and cries right after being fed, he may be feeling some sort of tummy pain. Many parents swear by over-the-counter anti-gas drops for babies or gripe water (made from herbs and sodium bicarbonate). Get your doctor's okay before using either of these.

Milestones

Even if your baby isn't colicky and has never been fussy after eating, an occasional bout of gas pain can make him miserable until he works it out. If you suspect gas, try something simple to eliminate it such as putting him on his back, holding his feet, and moving his legs in a gentle bicycling motion.

Discover other possible causes of babies abdominal pain, including reflux, stomach flu, milk allergy, lactose intolerance, constipation, and intestinal blockage.

7. Too cold or too hot

When your baby feels chilly, such as when you remove his clothes to change a diaper or clean his bottom with a cold wipe, he may protest by crying.

Newborns like to be bundled up and kept warm — but not too warm. As a rule, they're comfortable wearing one more layer than you need to be comfortable. Babies are less likely to complain about being too warm than about being too cold, and they won't cry about it as vigorously.

8. Something small

Babies can be troubled by something as hard to spot as a hair wrapped tightly around a tiny toe or finger, cutting off circulation. (Doctors call this painful situation a "hair tourniquet," and it's one of the first things they look for if a baby seems to be crying for no reason.)

Some babies are extra sensitive to things like scratchy clothing tags or fabric.And they can be very picky (understandably) about subtleties ranging from the position they're held in to the bottle you offer.

9. Teething

Teething can be painful as each new tooth pushes through tender young gums. Some babies suffer more than others, but all are likely to be fussy and tearful at some point along the way.

If your baby seems to be in pain and you're not sure why, try feeling his gums with your finger. You may be surprised to discover the hard nub of a baby tooth on its way in.

On average, the first tooth breaks through between 4 and 7 months, but it can happen earlier.

10. Wants less stimulation

Babies learn from the stimulation of the world around them, but sometimes they have ahard time processing it all — the lights, the noise, being passed from hand to hand. Crying can be a baby's way of saying, "I've had enough."

Many newborns enjoy being swaddled. It seems to make them feel more secure when the world gets overwhelming. If your baby's too old for swaddling or doesn't like it, try retreating to a serene spot and letting your baby vent for a while to manage a meltdown.

11. Wants more stimulation

A "demanding" baby may be outgoing and eager to see the world. And often the only way to stop the crying and fussing is to stay active. This can be exhausting for you!

Try "wearing" your baby in a sling, front carrier, or backpack

Plan plenty of activities. Hang out with other parents with babies. Go on regular outings to kid-friendly places, whether that's your local playground, a children's museum, or the zoo.

12. Not feeling well

If you've met your baby's basic needs and comforted him and he's still crying, he could be coming down with something. You may want to check his temperature to rule out a fever and be alert for other signs of illness.

The cry of a sick baby tends to be distinct from one caused by hunger or frustration. If your baby's crying "just doesn't sound right," trust your instincts and call or see a doctor.

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